VOL. III MARCH 1952 NO. 3

UNITED STATES ARMED FORCES MEDICAL JOURNAL

Published Monthly by the Armed Forces Medical Publication Agency, Department of Defense

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THE PRINTING OF THIS PUBLICATION HAS BEEN APPROVED BY THE DIRECTOR OF THE BUREAU OF THE BUDGET, OCT. 19, 1949

UNITED STATES
GOVERNMENT PRINTING OFFICE
WASHINGTON : 1952

For sale by the Superintendent of Documents, U. S. Government Printing Office
Washington 25, D. C.

Foreword

The UNITED STATES ARMED FORCES MEDICAL JOURNAL represents the unification of the BULLETIN OF THE UNITED STATES ARMY MEDICAL DEPARTMENT and the UNITED STATES NAVAL MEDICAL BULLETIN. This joint periodical is the medium for disseminating information of administrative and professional interest to all medical personnel of the Department of Defense.

The Chairman of the Armed Forces Medical Policy Council and the Surgeons General of the several services invite all medical officers, dental officers, Medical Service Corps officers, Nurse Corps officers, and officers of the Veterinary Corps of the Armed Forces, and the medical consultants of the Army, Navy, and Air Force to submit manuscripts for publication in this JOURNAL.

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OFFICE OF THE SECRETARY OF DEFENSE
ARMED FORCES MEDICAL POLICY COUNCIL
WASHINGTON 25, D.C.

MEMO: Personnel of the Medical Services of the United States Armed Forces

A basic responsibility of a medical officer is to make available to others any ideas he may have for bettering the medical services. One medium through which this responsibility can be discharged so far as medical equipment is concerned exists at Fort Totten, New York. Here is a tri-service laboratory which is equipped and staffed to translate these ideas into actual full-scale models for the purpose of developing equipment to be used by the medical services of the Army, Navy, and Air Force. Known as the Engineering Development Division, it operates under the administrative control of the Armed Services Medical Procurement Agency and the management control of the Armed Services Medical Materiel Standardization Committee.

Recently a tri-service team, comprised of medical supply personnel of the three military departments and representatives from the Engineering Development Division, visited medical installations in Korea and Japan to determine the adequacy of medical supplies and equipment in the Far East Command and to learn the ideas of those actually using medical field equipment as to how it may be improved. Because of the findings of this engineering group and the many excellent recommendations made by medical personnel in the field, even in the most forward areas, a number of projects will be undertaken by this laboratory either to develop new equipment or still further improve present equipment.

Every medical officer has the privilege and obligation of contributing to his medical service by suggesting improvements in the equipment which he is using. The suggestions should be submitted in writing with sketches or photographs if possible, through channels, to the Surgeon General of his respective service. The Surgeon General, after evaluating the suggestions, will recommend to the Engineering Development Division the projects to be undertaken.

W. Randolph Lovelace, II, M. D.
Chairman

Dextran Infusion in Normal Chinese Men[1]

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Joseph S. Chen, *M. S.*
C. H. Chiang, *M. D.*
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J. C. Yue, *M. B.*

DEXTRAN is a high-molecular-weight polysaccharide formed by the growth in culture of various micro-organisms, particularly that of the nonpathogenic *Leuconostoc mesenteroides*, in a substrate of sucrose and phosphate. The micro-organism produces an enzyme or enzymes which synthesize dextran from glucose molecules. It was first discovered by German workers who regarded it as a nuisance in sugar refining. Attempts to produce antibodies in rabbits revealed that dextran is nonantigenic. This led to extensive studies in Sweden as to the feasibility of its being used as a plasma substitute *(2-6)*. It has certain theoretical advantages over other protein or nonprotein colloids in that it is nonantigenic, free from acid radicals, and can presumably be metabolized in the body *(7)*. Reports from Sweden have shown promising results on its clinical use. Thorsen *(6)* observed 5 unfavorable reactions in 16,750 dextran-phosphate infusions. Tests in the United States, however, showed conflicting results. Turner et al. *(8)* reported that reactions were observed in 10 of 30 patients. On the

(1) From National Defense Medical Center, Taipeh, Taiwan, China.

(2) Grönwall, A., and Ingelman, B.: Untersuchungen über Dextran und sein Verhalten bei parenteraler Zufuhr. Acta physiol. Scandinav. 7: 97, 1944; 9: 1, 1945.

(3) Bohmansson, G.; Rosenkvist, H.; Thorsen, G.; and Wilander, O.: Clinical experiences with dextran as plasma substitute. Acta chir. Scandinav. 94: 149-167, 1946.

(4) Ingelman, B.: Dextran and its use as plasma substitute. Acta Chem. Scandinav. 1: 731, 1947.

(5) Grönwall, A., and Ingelman, B.: Dextran as substitute for plasma. Nature, London 155: 45, Jan. 13, 1945.

(6) Thorsen, G.: Recent investigations of properties of dextran. Nord. Med. 40: 2374, 1948. (Swedish text, summary in English.)

(7) Editorial: Dextran as plasma substitute. J. A. M. A. 139: 850-851, Mar. 26, 1949.

(8) Turner, F. P.; Butler, B. C.; Smith, M. E.; and Scudder, J.: Dextran: An experimental plasma substitute. Surg., Gynec. & Obst. 88: 661, 1949.

other hand Lundy et al. (9) administered dextran to 1,500 patients without untoward reactions. The reactions noted by various authors consisted of skin rash, urticaria, lumbar aching, asthmatic attacks, feeble pulse and fall of blood pressure, anxiety, and dyspnea, but it was generally agreed that the circulating volume in the cardiovascular system was well supported.

In most cases cited above, observations were made on surgical patients. It was thought that tests on normal persons might reveal more clearly the extent and nature of the reaction dextran could produce. This article deals with a study of dextran infusion in 90 normal men.

EXPERIMENTAL

The dextran used was a 6 percent solution in saline in bottles of 500 ml. known as "clinical dextran." Two lots were used. Ninety normal Chinese men, most of whom were medical students, were used for the test. Table 1 shows their age, weight, and height.

TABLE 1. *Age, weight, and height of experimental subjects*

Age (years)	Number	Weight (pound)	Number	Height (inches)	Number
19	5	96-99	2	58-59	2
20-29	78	100-119	49	60-64	43
30-39	6	120-139	35	65-69	43
40-42	1	140-156	4	70-71.5	2

After the volunteers rested in bed for from 30 to 60 minutes in a special ward previously prepared for them, a brief physical examination was performed on each. Readings of temperature (rectal), pulse rate, respiration, and blood pressure were recorded. An erythrocyte count, hemoglobin determination, sedimentation rate, and plasma protein determination were made. A urine specimen was obtained from each for routine examinations. Then 500 ml. of dextran solution were given to each subject at a rate of from 4 to 13 ml. per minute. The shortest duration of infusion was 40 minutes, the longest 130 minutes (average 70). In no instance was the infusion interrupted because of untoward reaction. The temperature, pulse rate, respiration, and blood pressure were taken at 1, 3, 6, 12, and 24 hours after the completion of the infusion; the erythrocyte count was repeated at 1, 3, 6, and 24 hours; hemoglobin was determined at 3 and 6 hours; sedimentation rates and plasma protein determinations were made 6 hours after infusion; and urine was collected at 6 and 24 hours.

Sahli tubes were used for hemoglobin determination. For sedimentation rates, Westergren tubes were used and observations were made for

(9) Lundy, J. S., and others: Annual report for 1947 of Section on Anesthesiology; including data and remarks concerning blood transfusion and use of blood substitutes. Proc. Staff Meet., Mayo Clin. 23: 432-445, Sept. 15, 1948; 460-462, Sept. 29, 1948.

1 hour. Plasma proteins were determined photometrically according to Greenberg's method *(10)*. The amount of dextran excreted in urine was determined by measuring reducing sugar after acid hydrolysis. The condition of full hydrolysis of dextran and least decomposition of glucose was previously worked out. To 0.25 ml. of urine, 1 ml. of twice normal HCl was added. The resulting mixture was then heated in a boiling water bath for 30 minutes to attain optimal hydrolysis. After cooling and neutralization the reducing sugar was estimated by the Folin-Wu method. All subjects were kept at bed rest and carefully watched for 24 hours. They were given full diet and fluids as desired. At the end of 24 hours they were discharged if no reaction occurred. Those with symptoms and elevation of temperature were kept under obser ation for another 24 hours until all symptoms subsided.

RESULT AND DISCUSSION

Reactions. Untoward reactions in the form of urticaria, asthmatic attacks, et cetera, as reported by Swedish and American workers, were not observed in the present series. Pyrogenic reactions, however, were encountered. Eighteen of the 90 men showed some elevation of rectal temperature above 99.6° F. (table 2). The elevations were *all* found to occur from 3 to 6 hours after the infusion. Of the 18 cases, only 4 complained of discomfort, which consisted of feverishness, dizziness, headache, and anorexia; and 7 showed some increase in the pulse rate. Case 84 had the most marked reaction among the group. He had mild chilly sensation shortly after infusion, followed by feverishness, dizziness, and headache. His erythrocyte sedimentation rate was increased from 6 to 30 mm. per hr. There were no significant changes in blood pressure and urine output, however. The other 3 cases (8, 10, and 83) showed much milder reactions. All 4 recovered within 24 hours. All the 18 subjects, with the exception of the first, gave low initial values either in hemoglobin content or in plasma protein level, or showed marked drop in either value after infusion. Subject 83 gave a history of splenomegaly 1 year preceding this study. What bearing, if any, these facts may have had on the occurrence of thermal reaction is unknown.

Temperature. With the exception of subjects 8, 10, 83, and 84 it is difficult to say whether the slight rise in rectal temperature was entirely due to dextran. Seven subjects showed an initial rectal temperature of 99.6° to 100° F. before dextran was given. It is known that some variations in rectal temperature occurs in healthy persons *(11)*. Therefore in the absence of subjective discomfort and an appreciable increase in the pulse rate, the reaction observed in the other 14 subjects was considered negligible. At the close of the experiment, 10 subjects (8, 10, 13, 51, 52, 55, 66, 70, 83, and 84) were selected from

(10) Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry. Vol. II: Methods. Williams & Wilkins Co., Baltimore, Md., 1932. p. 691.

(11) Best, C. H., and Taylor, N. B.: The Physiological Basis of Medical Practice. 4th edition. Williams & Wilkins Co., Baltimore, Md., 1945. p. 623.

TABLE 2. *Rectal temperature, pulse rate, sedimentation rate, hemoglobin, and plasma protein in 18 subjects showing elevated temperature after dextran infusion*

Subject	Type of observation	Before infusion	Hours after infusion					Subjective discomfort
			1	3	6	12	24	
1	Temperature (°F.)	99.4	99.6	99.2	100.6	98.2	98.3	None
	Pulse rate	76	84	82	76	68	72	
	Sedimentation rate	2			11			
	Hemoglobin (percent)	80			72			
	Plasma protein (gm. per 100 ml.)	7.4			6.66			
5	Temperature (°F.)	99.4	99.2	99.4	99.6	101.0	99.0	None
	Pulse rate	76	73	84	76	76	80	
	Sedimentation rate	4			9			
	Hemoglobin (percent)	70		70	64			
	Plasma protein (gm. per 100 ml.)	6.73			5.57			
8	Temperature (°F.)	99.6	99.4	99.8	100.6	99.8	100.4	Feverishness, transient
	Pulse rate	78	79	84	92	88	90	
	Sedimentation rate	3			6			
	Hemoglobin (percent)	84		80	62			
	Plasma protein (gm. per 100 ml.)	6.72			5.97			

TABLE 2. *Rectal temperature, pulse rate, sedimentation rate, hemoglobin, and plasma protein in 18 subjects showing elevated temperature after dextran infusion*—Continued

Subject	Type of observation	Before infusion	Hours after infusion					Subjective discomfort
			1	3	6	12	24	
10	Temperature (°F.)	99.0	98.7	98.9	100.0	101.0	99.6	Feverishness and dizziness 6 hours after infusion
	Pulse rate	64	60	72	80	96	90	
	Sedimentation rate	3			7			
	Hemoglobin (percent)	82		80	80			
	Plasma protein (gm. per 100 ml.)	5.43			5.44			
12	Temperature (°F.)	99.4	99.2	99.2	100.0	99.8	99.8	None
	Pulse rate	68	64	65	66	64	70	
	Sedimentation rate	2			4			
	Hemoglobin (percent)	76		80	80			
	Plasma protein (gm. per 100 ml.)	5.37			4.93			
13	Temperature (°F.)	100.0	99.8	99.8	100.4	99.4	99.6	None
	Pulse rate	76	74	78	80	70	88	
	Sedimentation rate	3			12			
	Hemoglobin (percent)	86		75	76			
	Plasma protein (gm. per 100 ml.)	6.14			5.67			

TABLE 2. *Rectal temperature, pulse rate, sedimentation rate, hemoglobin, and plasma protein in 18 subjects showing elevated temperature after dextran infusion*—Continued

Subject	Type of observation	Before infusion	Hours after infusion					Subjective discomfort
			1	3	6	12	24	
14	Temperature (°F.)	99.2	99.6	99.2	100.0	98.6	99.8	None
	Pulse rate	72	84	72	82	80	72	
	Sedimentation rate	2			4			
	Hemoglobin (percent)	80		70	68			
	Plasma protein (gm. per 100 ml.)	5.18			5.11			
42	Temperature (°F.)	98.6	98.6	99.5	100.0	99.6	98.8	None
	Pulse rate	70	72	75	80	64	80	
	Sedimentation rate	2			2			
	Hemoglobin (percent)	74		70	70			
	Plasma protein (gm. per 100 ml.)	7.87			6.47			
44	Temperature (°F.)	99.6	99.0	99.5	100.4	100.3	99.6	None
	Pulse rate	72	76	80	86	86	74	
	Sedimentation rate	3			6			
	Hemoglobin (percent)	72		75	56			
	Plasma protein (gm. per 100 ml.)	6.91			5.00			

TABLE 2. *Rectal temperature, pulse rate, sedimentation rate, hemoglobin, and plasma protein in 18 subjects showing elevated temperature after dextran infusion*—Continued

Subject	Type of observation	Before infusion	Hours after infusion					Subjective discomfort
			1	3	6	12	24	
51	Temperature (°F.)	99.6	99.3	99.3	100.4	99.4	98.0	None
	Pulse rate	74	69	83	86	84	76	
	Sedimentation rate	2			4			
	Hemoglobin (percent)	78		78	80			
	Plasma protein (gm. per 100 ml.)	7.11			4.90			
52	Temperature (°F.)	99.4	99.6	100.4	99.6	99.0	99.4	None
	Pulse rate	88	68	96	84	80	80	
	Sedimentation rate	1			5			
	Hemoglobin (percent)	84		80	78			
	Plasma protein (gm. per 100 ml.)	7.60			5.60			
55	Temperature (°F.)	98.6	99.3	100.0	99.0	98.6	99.2	None
	Pulse rate	72	72	72	72	52	68	
	Sedimentation rate	2			2			
	Hemoglobin (percent)	72		70	72			
	Plasma protein (gm. per 100 ml.)	7.56			5.98			

TABLE 2. *Rectal temperature, pulse rate, sedimentation rate, hemoglobin, and plasma protein in 18 subjects showing elevated temperature after dextran infusion*—Continued

Subject	Type of observation	Before infusion	Hours after infusion					Subjective discomfort
			1	3	6	12	24	
66	Temperature (°F.)	99.8	99.1	99.9	100.6	99.6	99.4	None
	Pulse rate	88	84	84	88	80	86	
	Sedimentation rate	3			8			
	Hemoglobin (percent)	72		70	64			
	Plasma protein (gm. per 100 ml.)	6.02			5.70			
70	Temperature (°F.)	100.0	100.1	99.8	100.6	99.6	100.2	None
	Pulse rate	72	62	80	65	60	68	
	Sedimentation rate	3			3			
	Hemoglobin (percent)	74		72	72			
	Plasma protein (gm. per 100 ml.)	6.52			5.92			
82	Temperature (°F.)	98.6	99.0	98.8	99.8	100.0	99.6	None
	Pulse rate	84	82	88	102	90	74	
	Sedimentation rate	8			26			
	Hemoglobin (percent)	70			64			
	Plasma protein (gm per 100 ml.)	6.66			5.86			

TABLE 2. *Rectal temperature, pulse rate, sedimentation rate, hemoglobin, and plasma protein in 18 subjects showing elevated temperature after dextran infusion*—Continued

Subject	Type of observation	Before infusion	Hours after infusion					Subjective discomfort
			1	3	6	12	24	
83	Temperature (°F.)	97.0	97.4	98.6	100.0	100.8	98.7	Anorexia and dizziness for 48 hours
	Pulse rate	76	72	84	92	88	72	
	Sedimentation rate	3			18			
	Hemoglobin (percent)	82			66			
	Plasma protein (gm. per 100 ml.)	6.21			5.79			
84	Temperature (°F.)	97.8	99.6	99.3	102.0	99.0	98.0	Slight chilliness, followed by fever, headache, and dizziness for 12 hours
	Pulse rate	78	86	96	88	84	84	
	Sedimentation rate	6			30			
	Hemoglobin (percent)	72			62			
	Plasma protein (gm. per 100 ml.)	6.40			6.34			
89	Temperature (°F.)	99.6	100.3	99.9	98.9	98.2	98.8	None
	Pulse rate	88	84	84	78	62	64	
	Sedimentation rate	6			13			
	Hemoglobin (percent)	76			64			
	Plasma protein (gm. per 100 ml.)	7.40			5.80			

the group of 18 with temperature elevation to have their 24-hour temperature checked under the same conditions but without dextran infusion. Two (52 and 55) were given 500-cc. normal saline infusion. In none was any elevation of temperature observed.

Blood pressure. In 60 percent of the subjects no change of blood pressure was observed incident to dextran infusion. An elevation of from 10 to 20 mm. Hg. was noted in the other 40 percent from 1 to 6 hours after the infusion. This elevation appeared to be within the range of normal fluctuation rather than the effect of dextran.

Sedimentation rate. The average rate in 89 subjects (1 undetermined) was 3.38 with a standard deviation of ± 2.83. Except in 7 subjects whose rate 6 hours after infusion showed a decrease, the rest showed an average increase of 3.42 mm. per hr. with a standard deviation of ± 4.94. The increases varied from none to as much as 24 mm. per hr. Most subjects had readings that were twice the original values. Bull et al. *(12)* stated that the sedimentation rate increases in proportion to the increase of the dextran level in the blood. Because readings were taken only once before and after infusion in the present study, we can only say that dextran did cause some increase of the sedimentation rate.

TABLE 3. *Mean values (with standard deviation) of erythrocyte count, hemoglobin, and sedimentation rate in 90 men before and after dextran infusion*

	Before infusion	Hours after infusion		
		3	6	24
Erythrocyte count (million)	3.83 ± 0.33	3.45 ± 0.36	3.16 ± 0.53	3.80 ± 0.37
Hemoglobin (percent)	77.50 ± 4.64	74.50 ± 4.37	67.70 ± 4.30	
Sedimentation rate	3.38 ± 2.83		7.30 ± 5.34	

Erythrocyte count and hemoglobin. Studies by Thorsen *(13)* on 8 medical students showed a drop of about 10 percent in the hemoglobin, erythrocyte count, and the hematocrit readings. The drop was followed by restitution to or over the original level 1 hour later, and there was a second drop within the next 8 hours which lasted for more than 24 hours. In the present study erythrocyte counts were followed for only 24 hours and hemoglobin was determined at 3 and 6 hours. Our findings did not confirm the observations of Thorsen. The diluting effect disappeared at the end of 24 hours as shown in table 3. The erythrocyte count in the present series dropped an average of about 20 percent, the drop being most marked 6 hours after infusion in most subjects. The

(12) Bull, J. P.; Ricketts, C.; Squire, J. R.; Maycock, W. d'A.; Spooner, S. J. L.; Mollison, P. L.; and Paterson, J. C. S.: Dextran as plasma substitute. Lancet 1: 134-143, Jan. 22, 1949.

(13) Thorsen, G.: On dextran. Lecture given before the Swedish Medical Society, May 24, 1949.

drop in red blood cell counts was more marked in our series than in those of Thorsen, and is perhaps due to the slight degree of anemia commonly found among normal Chinese adults *(14)*.

TABLE 4. *Mean change (with standard deviation) of plasma protein level*

Determination	Before infusion	6 hours after infusion
Plasma protein (gm. per 100 ml.)	6.45 ± 0.71	5.99 ± 0.61
Albumin (gm. per 100 ml.)	4.04 ± 0.38	3.72 ± 0.42
Globulin (gm. per 100 ml.)	2.32 ± 0.48	2.27 ± 0.16
A/G ratio	1.74 ± 0.53	1.63 ± 0.60

Plasma protein and A/G ratio. Thorsen *(15)* reported that the plasma protein level was reduced after dextran infusion. The reduction reached a maximum 6 hours after infusion and was followed by gradual recovery. A plasma protein value of as low as 2 gm. per 100 ml. was observed. A fall of plasma protein level was also observed in the present series. Except in 7 subjects whose plasma protein levels were not lowered by dextran infusion, the average values were 6.45 gm. per 100 ml. and 5.99 gm. per 100 ml. before and 6 hours after infusion respectively. The lowest value observed (subject 46) was 4.48 gm. per 100 ml. (7.4 gm. per 100 ml. before infusion) which agrees closely with the values reported by Bull et al. *(12)*. The A/G ratio decreased from an average of 1.74 to 1.63. This slight change in A/G ratio confirms Thorsen's *(15)* finding that the reduction is greater in the albumin than the globulin fraction. It is our opinion that the fall of plasma protein level after dextran infusion was due more to the administration of fluid than to that of dextran. The change of A/G ratio, on the other hand, could be mainly effected by dextran. The results are shown in table 4.

Renal excretion of dextran. Twenty-four hour urine samples were collected from all 90 subjects at 6 and 24 hours after dextran infusion. The amount of dextran recovered in each period varied greatly. From table 5 it can be seen that the amount of dextran excreted in the first 6 hours after infusion varied in extreme cases from 2.5 times lower to 5.5 times higher than the amount excreted in the next 18 hours, the average being about 1.5 times higher. Bull et al. *(12)* reported that the amount of dextran excreted by 7 subjects in from 2 to 5 days varied from 9 to 26 percent. They reported on 1 patient with carcinoma who was infused with 30 gm. of dextran, and the amount excreted in 0 to 8, 9 to 16, and 17 to 24 hours was found to be 3.8, 1, and 1.2 gm., respectively. Bloom and Willcox *(16)* reported on 2 subjects

(14) Wan, S.: Health and nutrition in Chinese Army. Chinese J. Nutrition. 2: 45, 1947.
(15) Thorsen, G.: Dextran as a plasma substitute. Lancet. 1: 132-134, Jan. 22, 1949.
(16) Bloom, W. L., and Willcox, M. L.: Determination of dextran in blood and urine. Proc. Soc. Exper. Biol. & Med. 76: 3-4, Jan. 1951.

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into each of whom 30 gm. of dextran was infused, and the amount excreted in 24 hours was found to be 6.4 and 7.5 gm. Because individual variations are marked and the molecular size of the dextran used probably varies from batch to batch, data on renal excretion as obtained by various authors cannot be accurately compared.

TABLE 5. *Renal excretion of dextran after infusion of 30 gm. of clinical dextran in 500 ml. of saline solution*

	Dextran excreted					
	After 6 hours		Next 18 hours		Total in 24 hours	
	Grams	Percent	Grams	Percent	Grams	Percent
Lowest	0.50	1.66	0.11	0.38	0.61	2.04
Mean	1.05	3.49	0.71	2.37	1.76	5.87
Highest	2.90	9.67	1.17	3.89	4.07	13.56
Standard deviation of mean	0.45	1.46	0.43	1.40	0.64	2.08

SUMMARY

Dextran infusions were given to 90 normal Chinese men. Each received 500 ml. of a 6 percent solution of clinical dextran intravenously. No skin reactions, asthmatic attacks or circulatory reactions were encountered. Mild pyrogenic reactions were observed in 4 subjects. Another 14 had a slight elevation of rectal temperature of no clinical significance. A drop in erythrocyte count and hemoglobin percentage and an increase in the sedimentation rate were observed after dextran infusion, the effect being most marked 6 hours after infusion. Erythrocyte counts returned to normal at the end of 24 hours. A fall of plasma protein level after dextran infusion was observed in most of the subjects. The fall was probably more related to fluid administration than to dextran. The decrease in A/G ratio after infusion was thought to be more directly related to dextran. Renal excretion of dextran was marked during the first 6 hours after infusion. Individual variations were marked in the amounts excreted.

NOTE: In the course of this study 2 emergency cases (multiple compound fracture and gastric hemorrhage) were given dextran infusion immediately on admission to the hospital. Both patients recovered. There was little doubt that the infusions helped to save these lives although no detailed studies were made.

ACKNOWLEDGMENT: Grateful acknowledgment is made to the American Bureau for Medical Aid to China which obtained the dextran and transported it by air to Taiwan and made the grant under which the experimental work here reported was carried out.

Delayed Primary Closures in War Wounds

Carl A. Broaddus, Jr., *Lieutenant, MC, U. S. N.*

WHILE medical officer in charge of the flesh wounds ward aboard the hospital ship U. S. S. *Haven* in Korea from 24 April to 7 October 1951, I had the opportunity to make the following observations relative to delayed primary wound closures. Two different technics of closure and postoperative management were used. In order to restrict coverage to these 2 technics, I shall discuss only patients that I operated on and, of these, only those who could be followed through to healing or failure. At times of mass embarkations, rapid evacuation of patients to Japan was necessary in order to keep beds available. This precluded postoperative evaluation of many patients.

During this time there were 56 patients (82 sutured wounds) who could be followed adequately. These patients came to us either directly from combat or via one or more field hospitals and on arrival their wounds were usually from 1 to 7 days old. Some had been adequately débrided, some only minimally (skin-edges trimmed but little more), and others had received only a battle dressing. They had almost invariably received daily injections of 300,000 units of procaine penicillin and a tetanus toxoid booster.

Débridement and management prior to delayed closure. In the treatment of these patients, many textbook precepts could not be followed, especially as regards débridement of all or nearly all war wounds. A man admitted with a fairly clean 4-day-old wound had nothing to gain from débridement. Instead, continuous daytime compresses of 0.5 percent acetic acid were applied to the wound (through a bulky dressing), and the dressing was changed every second day until the wound was ready for suturing, usually from 3 to 10 days later.

On the other hand, a badly contaminated wound, or a wound associated with swelling, discoloration, or gas in the tissues, would be débrided regardless of the age of the wound. Following débridement, unless the wound was over 13 cm. long, it was left open with Penrose

drains in any deep recesses and a bulky dressing was applied. Two days postoperatively the dressing was changed and the drains removed or shortened. Thereafter, acetic acid compresses were applied.

In patients in whom the débrided wound was fairly shallow as compared to the size of the skin defect, no drain was used and the wound was packed loosely with fluffed 4-by 4-inch dressings. A bulky dressing was then applied and treatment with acetic acid compresses was started on the same day. In general, therefore, if the acetic acid could be expected to reach the base of the wound, drains would be omitted because prompt institution of acetic acid compresses was found to shorten the latent period between débridement and closure. Patients experienced no pain on removal of these moist packs in which blood and serum were never given a chance to coagulate. This is in contrast to the wounds that we received which had been débrided elsewhere and in which there had been no chance to keep the dressings moist. The removal of these dressings was always painful even when preliminary moistening was carried out.

Ambulatory and semiambulatory patients were made responsible for keeping their own dressings wet at all times during the day. At night they were allowed to discontinue moistening the dressings. The 0.5 percent acetic acid was kept on the ward in a bottle with a rubber tube and stopcock for this purpose.

Wounds containing foreign bodies. The treatment of wounds containing radiopaque foreign bodies did not differ greatly from the treatment of other wounds similar in age and appearance, unless the foreign body was located in a joint or elsewhere so that incapacity might result from its presence. These foreign bodies were removed, preferably under spinal or general anesthesia, either through a surgical incision or, if practicable, through the wound of entry. All of these foreign bodies were located. In wounds requiring débridement any retained foreign body would be sought, though in the occasional patient in whom it could not be found, only surgical pride was believed to have suffered and the procedure was not protracted. Foreign bodies palpable beneath the skin were removed unless very small, either separately under local anesthesia or at the time of the débridement of the wound of entry.

General ward care of these patients consisted of: (1) giving 300,000 units of procaine penicillin daily until healing was complete, (2) giving 0.5 cc. of tetanus toxoid on admission, regardless of previous doses, (3) applying acetic acid compresses, (4) giving whole blood as indicated, and (5) giving 200 mg. of ascorbic acid daily from the day of wound closure until the sutures were removed.

DELAYED PRIMARY CLOSURE: TWO TECHNICS

All principles of treatment so far mentioned were standard throughout the period covered by this report. The delayed primary closures themselves, however, were performed in 2 different manners.

Classical closure by layers, with drainage (group 1). The first 32 wounds (in 22 patients) were closed employing the usually accepted technic for delayed closures. Each wound was excised in so far as practicable; bleeders were tied meticulously with No. 00 plain catgut; the wound was irrigated with saline solution (warm if available) and closed in layers using No. 0 chromic catgut for the deep fascia, No. 00 plain catgut elsewhere beneath the skin, and cotton or dermalon interrupted vertical mattress sutures for the skin. A small rubber dam (slip) drain was left protruding from the most dependent portion of the wound, except in face wounds, and was removed 2 days postoperatively. Cotton elastic bandages were used only occasionally, most dressings being held in place as snugly as possible with adhesive tape. Sutures were removed on the sixth, seventh, or eighth postoperative day, frequently half on one day and half on the next. Patients with leg and thigh wounds were allowed to be up and around minimally and were allowed bathroom privileges.

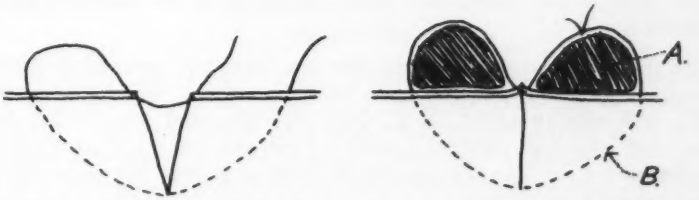

Figure 1. Technic of one-layer wire closure of war wounds. (A) *4- by 4-inch dressings rolled tightly longitudinally.* (B) *No. 009 steel wire suture extending to base of wound.*

Wire closure in one layer, without drainage (group 2). Early in July I was introduced by Commander S. H. Tolins, MC, U. S. N., to a new technic to which I have adhered with few changes. This technic was applied to 32 patients (50 wounds) who could be adequately followed. Following the usual excision of the wound, with clamping of bleeders, the wound was packed for a few minutes with warm (120° F.) saline sponges. Clamps were then removed and no ties employed except on unusually large tissue bleeders which were tied with No. 0000 chromic catgut using about 2 such ligatures in every 3 wounds. Closure was effected with deeply biting vertical mattress sutures of No. 009 steel wire. The deep outside bite of each suture entered the skin from 2 to 2.5 cm. from the edge of the defect, traversed the base of the wound, and emerged similarly on the opposite side. The return throw of the suture caught only the skin edges. The sutures were all placed before tying and then tied over tightly rolled 4- by 4-inch dressings, one on each side of the wound (fig. 1). This prevented cutting the skin, provided a tamponading effect, and tended to evert the skin edges slightly. Although the wire sutures were tied only tightly enough for good skin approximation, no bleeding was noted after they were tied, and no hema-

tomas were encountered. The skin edges were examined between the gauze rolls to see that no loose edges of the gauze were dipping into the wound and that the skin edges were accurately approximated. An occasional skin-edge suture of No. 000 dermalon was necessary midway between the wire sutures, which were placed about 2 cm. apart. A third rolled 4-by 4-inch dressing was tied into the free ends of the previously tied wire sutures and a dry sterile dressing applied. In addition a cotton elastic bandage was applied snugly to wounds of an extremity, shoulder, or buttocks from above the wound to the end of the involved part and left in place for 6 or 7 days (8 or 9 days for wounds below the knee or in wounds in which there was undue tension). In most thigh wounds and in all wounds of the elbow, wrist, knee, or ankle immobilization was insured by incorporating a molded plaster splint in the dressing. An airplane splint was used in wounds over the deltoid muscles or upper scapula. In trunk wounds, a cotton elastic bandage was left on for about 2 days before being removed for the sake of comfort. Strict immobilization of the part was demanded and patients with lower extremity wounds were kept in bed except for being allowed to go to the bathroom on crutches.

In an occasional wound, especially one about the forehead or smaller wounds elsewhere, No. 000 nylon or dermalon was substituted for the No. 009 wire (wounds 28, 42, 53a, 25b, 27a, and 49b). A No. 005 tantalum wire was used in 2 wounds (44b and 50a). Results were the same as for the No. 009 steel wire, and no further reference will be made to these variations.

RESULTS

Tables 1 and 2 show the results in wounds sutured by the 2 major technics discussed in this report. Wounds were not considered healed until 24 hours after removal of the sutures, at which time any separation of the wound edges or drainage was noted. Separations which healed spontaneously within 6 days, with or without acetic acid compresses, were classed as minor; those healing within 12 days, moderate; and those persisting beyond 12 days or requiring resuture, severe. Of the 32 wounds sutured in group 1, there were 8 separations, including 2 minor, 3 moderate, and 3 severe (one of which eventually required skin grafting and was healed 53 days after the initial sutures were removed). These 8 separations represented 25 percent of the wounds sutured by this technic.

Of the 50 wounds treated with the one-layer wire closure technic there were 4 separations (2 minor and 2 severe, one of which was not healed when the patient was transferred to another facility 9 days after the sutures were removed). It was estimated that healing would require 10 days more. The end of one of the gauze rolls had slipped between the edges of this wound and was not noticed at the time of operation. This was checked for in all subsequent operations. The other was resutured 5 days after removal of the sutures and was healed 12 days

after the original sutures were removed. A bullet was removed from this wound, the wound excised, and closure performed at the same operation. Results following this type of operation were poor with either of the technics described. The 4 separations in this group represented only 8 percent of the wounds thus treated.

A total of 107 days were lost because of wound separations in the 32 wounds of group 1, or 3.34 days per wound, although if the patient with a very ischemic lower leg wound above the medial malleolus of the tibia is omitted, this figure is reduced to 55 days lost or 1.77 days per wound.

In the 50 wounds of group 2, there was a total of 34 days, or 0.68 day per wound, lost beyond the expected day of healing. Here, too, if the patient in whom the gauze was allowed to remain between the edges of the wound is omitted, 16 days, or 0.33 day per wound, were lost. Using either the inclusive or the exclusive figures, this represents only one-fifth as great a loss of time beyond the expected day of healing as for the wounds of group 1 (table 3).

DISCUSSION

This comparison assumes even greater significance in view of the fact that the patients of group 2 included the more severe wounds. Because of the rapid influx of patients before July, a more rapid turnover was mandatory. This necessitated evacuation of the more severely wounded patients who would otherwise have been kept after July and followed through to healing. Also, after the first few closures using wire, I found that large avulsions could be sutured by this technic and, even though under tension, all these wounds healed perfectly. I would previously have employed grafts on at least 4 of the patients with avulsion on whom I used wire and obtained excellent results despite tension.

The possibility of postoperative herniations with the wire-closure technic. I have been asked whether this one-layer type of closure might not predispose to herniations through fascial layers which are not individually sutured. I do not believe so. While placing sutures, it is wise to pull any major fascial layer, such as the fascia lata, toward the center of the wound, thus allowing a large fascial bite to be taken on each side of the wound. This should result in adequate approximation. If further experience proves this incorrect, I see no objection to suturing the fascia with a few wire sutures, *after* the placing of the deeply biting sutures but before they are tied. This would be similar to the treatment of wounds too deep for skin sutures to reach the bottom. I encountered 2 such wounds, both of the buttock, and closed them with buried wire sutures out to the point where deep vertical mattress sutures would reach. Healing was uneventful. Although no drains were employed after adoption of the wire technic, there were no infections and no retained blood or effusion.

TABLE 1. *Results of closure by layers with drainage*

Type of management	Patient	Wound site	Time from débridement to closure (days)	Postoperative day sutures were removed	Postoperative day wound was healed	Time from admission to healing (days)	Time from wounding to healing (days)
Débridement with closure at same time	8a	Face	0	3	4	4	5
	8b	Face	0	3	4	4	5
	8c	Face	0	3	4	4	5
Débridement with or without removal of foreign body, closure later	1	Thigh	6	7	20(2)	23	27
	2a(1)	Arm	7	7	8	12	15
	2b	Arm	7	7	8	12	15
	3	Foot	5	6	7	13	16
	4a	Thigh	7	7	8	15	17
	4b	Thigh	7	7	8	15	17
	5	Forearm	8	6	7	16	18
	6	Leg	10	7	21(2)	32	35
	7	Arm	5	7	8	14	16
	12a	Arm	6	7	8	12	14
	12b	Arm	6	7	8	12	14
	12c	Buttock	6	7	8	12	14
Acetic acid compresses without débridement, delayed closure later	11a	Buttock		5	6	16	17
	11b	Buttock		5	6	16	17
	16a	Thigh		7	8	11	14
	16b	Thigh		7	8	11	14
	20	Thigh		7	9	13	20
Débridement with removal of foreign body and closure in same stage	13	Neck	0	4	13(2)	14	17
	14	Leg	0	7	11(2)	14	16
	15a(3)	Leg	0	6	7	8	12
	15b	Leg	0	6	7	8	12
	16c	Arm	0	7	14(2)	15	18

TABLE 1. *Results of closure by layers with drainage*—Continued

Type of management	Patient	Wound site	Time from débridement to closure (days)	Postoperative day sutures were removed	Postoperative day wound was healed	Time from admission to healing (days)	Time from wounding to healing (days)
Excision of foreign body, partial closure, final closure later	17	Leg	8	4	13(2)	23	28
	18	Leg	6	6	7	17	24
	19	Buttock	8	7	8	18	20
	22	Arm	8	9	10	20	26
	10	Leg	17	9	Needed reoperation (2) (4)		
Acetic acid compresses, later suture of previously dehisced wound	9	Thigh	13(5)	7	12(2)	18	44
	10	Leg	29(5)		24	70	72
	21	Thigh	15(5)	9	10	29	31
		Total	127	203	304	521	635
		Number wounds	17	32	32	32	32
		Average	7.47	6.34	9.50	16.3	19.8

(1) This patient needed physiotherapy to elbow for 11 days following wound healing. Function was but little improved so he was evacuated to Japan.

(2) These wounds were not satisfactorily healed 1 day after the removal of sutures.

(3) This patient needed physiotherapy for 5 days following wound healing to overcome slight foot drop.

(4) This man had a small-arms wound 2 inches above the medial malleolus of the tibia. The wound was débrided and partially closed on day of admission. Seventeen days later, because the small defect had made no progress toward healing, delayed closure was attempted. This failed and subsequent grafting was necessary. The area of the wound was markedly ischemic, and closure should probably not have been attempted.

(5) These figures represent number of days from removal of suture of previous, unhealed, delayed closure to this resuture, and are not included in the total or average of this column.

TABLE 2. *Results of wire closure in one layer without drainage*

Type of management	Patient	Wound site	Time from débridement to closure (days)	Postoperative day sutures were removed	Postoperative day wound was healed	Time from admission to healing (days)	Time from wounding to healing (days)	Splint
Débridement with or without removal of foreign body, closure later date	24	Chest	9	8	9	18	21	
	26(1)	Forearm	12	8	9	12	21	P
	28(2)	Chest	11	7	8	13	19	
	30	Thigh	5	7	12(3)	15	18	
	31	Back	23	8	9	16	32	
	32a	Leg	8	8	9	14	17	
	32b	Leg	8	8	9	14	17	P
	32c	Foot	8	8	26(3) (4)	31(4)	34(4)	
	33	Thigh	6	8	9	14	15	
	34(5)	Arm	6	8	9	14	15	A
	37	Back	8	7	8	18	22	A
	39	Buttocks	7	7	8	18	21	
	40a	Arm	5	7	8	11	13	
	40b	Arm	5	7	8	11	13	
	42(2)	Knee	5	6	7	10	12	P
	45	Thigh	8	7	8	14	16	P
	48	Leg	8	8	9	15	17	
	49a	Thigh	5	7	8	14	16	
	50a(6)	Elbow	4	7	8	11	14	P
	50b	Leg	4	7	8	11	14	P
	51	Thigh	5	7	8	14	18	
	52a	Neck	8	7	8	14	16	
	52b	Wrist	8	7	8	14	16	
	53a(2)	Arm	2	7	8	14	16	
	54a	Chest	6	7	8	16	17	
	55	Thigh	5	6	7	15	16	P

TABLE 2. *Results of wire closure in one layer without drainage*—Continued

Type of management	Patient	Wound site	Time from débridement to closure (days)	Postoperative day sutures were removed	Postoperative day wound was healed	Time from admission to healing (days)	Time from wounding to healing (days)	Splint
Acetic acid compresses without débridement, delayed closure later	25a(2) (7)	Arm		8	9	12	16	P
	25b(2)	Arm		8	9	12	16	P
	27a(2) (8)	Forearm		7	8	11	18	P
	27b	Thigh		7	8	11	18	
	29	Thigh		7	9(2)	16	23	
	35	Thigh		7	8	11	12	
	41a	Knee		7	8	11	15	
	41b	Thigh		7	8	11	15	
	41c	Thigh		7	8	11	15	
	41d	Forearm		7	8	11	15	
	41e	Forearm		7	8	11	15	
	44a	Abdomen		6	7	11	13	
	44b(6)	Abdomen		6	7	11	13	
	46	Neck		7	8	14	21	
	49b(2)	Face		3	4	8	10	
	53b(9)	Knee		7	8	14	17	
	53c	Knee		7	8	14	17	
	54b	Arm		7	8	16	17	
Débridement with removal of foreign body on closure in same stage	47	Thigh	0	7	Not healed (2)			
	43	Thigh	0	7	8	9	11	
Excision of foreign body, partial closure, final closure later	23(10)	Thigh	15	7	9	24	26	
	36	Buttock	11	7	8	20	21	

TABLE 2. *Results of wire closure in one layer without drainage*—Continued

Type of management	Patient	Wound site	Time from débridement to closure (days)	Postoperative day sutures were removed	Postoperative day wound was healed	Time from admission to healing (days)	Time from wounding to healing (days)	Splint
Acetic acid compresses, later suture of previously dehisced wound	38	Thigh	8(11)	8	9	13	29	P
	47	Thigh	3(11)	8	9	26	29	
		Total	215	355	420	689	868	
		Number wounds	30	50	49	49	49	
		Average	7.20	7.10	8.57	14.06	17.71	

P = Posterior plaster splint applied after suture.
A = Airplane splint applied after suture.
(1) This patient needed physiotherapy to forearm for 3 days following wound healing.
(2) 000 dermalon or nylon was used in place of steel wire.
(3) These wounds were not satisfactorily healed 1 day after removal of sutures.
(4) This patient had to be transferred to another facility 21 days after admission, 16 days after closure and figures are only approximate.
(5) This patient needed physiotherapy to arm for 5 days following wound healing.
(6) 005 tantalum wire was used in place of steel wire.
(7) This patient needed physiotherapy to knee for 12 days following wound healing (because of wounds not listed here).
(8) This patient needed physiotherapy to thigh for 6 days following wound healing.
(9) This patient needed physiotherapy to knee for 6 days following wound healing.
(10) This patient needed physiotherapy to thigh for 10 days following wound healing.
(11) These figures represent number of days from removal of suture of previous unhealed delayed closure to this resuture, and are not included in the total or average of this column.

TABLE 3. *Comparison of the results of treatment in groups 1 and 2*

Group	Number of wounds	Separations		Time lost because of separations (days)	Time lost (corrected figure) (days)	Time lost per wound (days)	Average time from expected healing to actual healing (days)	Average time from admission to healing (days)
		Number	Percent					
1	32	8	25	107	55	1.77	2.16	16.30
2	50	4	8	34	16	0.33	0.47	14.06

Foreign body removal with delayed closure, one stage. Uniformly poor results were obtained in wounds that were closed at the same operation in which a foreign body was removed. Of the 32 wounds in group 1, only 5 were treated in this manner. Yet of the 8 wound separations in this group, 3 came from those treated in this way. Similarly, of the 50 wounds in group 2, there were only 2 in which suture was performed at the time the foreign body was removed. One of these separated. This was the only separation encountered among the wounds of this group that required resuture. Combining the 2 groups, it is seen that of 12 total wound dehiscences in 82 wounds, 4 came from a total of only 7 wounds sutured at the time the foreign body was removed despite what was considered adequate excision of selected wounds and irrigation.

Return to duty. All the patients included in these 2 groups were not necessarily returned to duty as soon as their wounds were completely healed. Eight patients (2 from group 1 and 6 from the more severe wounds included in group 2) were held over for physiotherapy; 5 of these were returned to duty within 6 days after healing and 2 more within 12 days. One had to be evacuated to Japan because the continued stiffness in his wounded arm was not improved after 11 days of physiotherapy. Six other patients were held over for eye refractions and completion of treatment of ruptured eardrums. All others were returned to duty with the next duty party after completion of healing.

Cosmetic results in wounds closed without tension were equal in the 2 groups of patients, depending not on the technic but on the accuracy of apposition. In wounds closed under tension the cosmetic results obtained by the wire technic were superior because of the absence of cross-hatching. In the one forehead wound sutured by the wire technic, a 4 by 3 cm. avulsion was closed and the sutures were removed 3 days later. The patient was followed for 9 days after removal of the suture while a thigh wound was undergoing débridement and subsequent closure. The resulting forehead scar was barely perceptible on discharge.

Removal of sutures. Sutures were left in place on an average of 6.34 days in group 1, as compared to 7.10 days in group 2. This difference was due in part to 3 face wounds (8a, b, and c) being included in group 1, from all of which the sutures were removed in 3 days, and to the removal of sutures from 2 other wounds at the end of 4 days because of the presence of infection, whereas in group 2 there was only one face (forehead) wound and no sutures were removed ahead of schedule. Omitting these wounds from both groups, the average was 6.89 days for group 1 and 7.18 days for group 2.

The *average healing time* after closure was 9.50 days in group 1 and 8.57 days in group 2, or 3.16 days after removal of the sutures in group 1 and 1.47 days in group 2. Since no wounds were considered healed until 1 day after the sutures were removed, this means a prolongation of healing of 2.16 and 0.47 days respectively for the two groups.

The *time in the hospital* until the wound was healed was 16.3 days for group 1 and 14.06 for group 2, or 19.8 and 17.7 days respectively from wounding to healing.

Latent period between débridement and closure. The average period between débridement and suture was 7.47 days in group 1 and 7.20 days in group 2.

SUMMARY

The results obtained from delayed closures in group 2 were significantly better than those of group 1. The principal differences in treatment of the two groups therefore must be considered responsible. These differences were: (1) no buried sutures and an average of less than 1 buried ligature in the wounds of group 2; (2) smaller ligatures (No. 0000 chromic catgut rather than No. 00 plain catgut) were used in group 2; (3) one-layer rather than multilayer closures were used in group 2; (4) there was probably a reduced chance of tissue strangulation in group 2 because a through-and-through suture that merely approximates skin should also merely approximate deeper tissues, thus avoiding the common tendency to tie buried sutures too tightly; (5) after institution of the wire technic, warm saline solution was demanded for every wound whereas previously it had been used only when available (perhaps 30 percent of the time); (6) after adoption of the wire technic, complete immobilization of the sutured part was maintained whereas only decreased activity had been required of group 1 (11 plaster splints and 2 airplane splints were used in group 2 and none in group 1); (7) there may be less likelihood of fluids collecting in the one-layer closures (*1*); and (8) the possibility of improving surgical acuity having played an important part in the results is improbable because the improvement in results was not gradual but came about with the change of technic.

CONCLUSION

The following factors appeared to play a major role in effecting an improvement in the results following delayed primary closures observed with the adoption of the one-layer wire technic.

1. Thorough irrigation of excised wounds with *warm* saline solution prior to closure.

2. Reduction of the amount and caliber of absorbable ligatures and of porous sutures such as cotton or silk, especially buried sutures. This reduction of ligature material and elimination of porous sutures is accomplished by the one-layer wire closure and is the principal goal of this technic.

(*1*) Despite the infrequent ligation of bleeders in these wounds, the combination of inward pressure from the gauze "rolls" tied beneath the free loops of the vertical mattress sutures and the outward pull of the deep limb of the same sutures (through the base of the wound) should, theoretically at least, provide an excellent tamponading effect. In practice this seemed to be the case.

3. Complete immobilization of sutured wounds until removal of the sutures, using splints if necessary.

Delayed primary closures of wounds should *not* be performed at the time of removal of foreign bodies because of the high incidence of wound separations in these patients.

BOOK REVIEW

Measurement and Evaluation in Physical, Health, and Recreation Education, by *Leonard A. Larson,* B. A., B. P. E., M. Ed., Ph. D., Professor of Education, New York University, and *Rachael Dunaven Yocom,* B. A., M. A., Ph. D., Instructor in Education, New York University. 507 pages; illustrated. The C. V. Mosby Co., St. Louis, Mo., publisher, 1951. Price $7.50.

This is a comprehensive text dealing with the plan of measurement and evaluation in programs of physical education, health education, and recreation in relationship to the general field of education. Measurement is conceived as pertaining to those technics which furnish information about the *product* (the individual or group) of educational activity. Evaluation, on the other hand, is presented as concerning the *process* used in achieving results. According to the authors, a measurement and evaluation program should measure the product of the programs in physical, health, and recreational education and evaluate the process by which the results were attained.

This text comprises 5 sections, the first of which is devoted to a development of the basic philosophy. Section 2 covers the measurement of organic functions (nonlaboratory technics), motor skills, knowledge and attitudes, and individual adjustment. This latter factor includes the mental, emotional, and social aspects in addition to the physical. The third section presents the place of evaluation. Section 4 concerns analysis of program results, and the final section deals with administration.

Mention should be made of the use in this volume of "Photocode," a method for presenting pictorial illustrations in conjunction with step-by-step directions to simplify for the reader the various procedures in test administrations and statistical calculations. This text was written for use in both elementary and advanced courses in measurement and evaluation. Selected references supplement each chapter and permit further study on any particular problem.

—*Lt. Col. J. B. Parsons, U. S. A. F. (MSC)*

Use of Plasma Expanders in Korea[1]

IN OCTOBER 1951, when Dr. W. Randolph Lovelace, II, Chairman of the Armed Forces Medical Policy Council, was in Korea on the first hop of his round-the-world trip to inspect U. S. military installations, it was pointed out to him that during winter operations in Korea, the medical aid men in forward combat areas had great difficulty in giving plasma because the diluent froze before it could be added to the dried plasma or, if the diluent had been kept from freezing, as soon as it was added to the plasma and exposed to the low temperature, the solution froze. This had been called to the Council's attention before Dr. Lovelace's departure from the United States and he was particularly interested in confirming the reports.

On his return Dr. Lovelace referred the problem to the National Research Council. He realized, however, that it probably would not be solved in time to be of any value in the winter just passed, because of the amount of research necessary, and that alternatives must be explored for a temporary solution. Discussions were held with representatives of the Defense Department's Committee on Whole Blood, Blood Derivatives and Plasma Expanders, which included not only military specialists but representatives of the National Research Council, the National Institutes of Health, and other interested agencies. It was determined that the amount of alcohol, salines, or carbohydrates which could be used safely in a diluent for the dried plasma would, at the most reduce the freezing point only 2° or 3° F. The use of concentrated human serum albumin (of which a large stock was also on hand) instead of plasma as an emergency measure presented the only rational solution for the immediate future, and one which may prove the most satisfactory even after extensive research. Instructions for its use were issued to the military departments.

Although concentrated serum albumin also has a freezing point within the 28° to 30° F. range, it is stored in small enough volume (25 grams in 100 cc. solution) that several ampules can be carried in the clothing of medical aid men operating in forward combat areas and in that way it can be protected from freezing. Furthermore, it does not have to be reconstituted prior to its use and can be injected rapidly before freezing

(1) From the Armed Forces Medical Policy Council.

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occurs. It combats shock most satisfactorily when there is little or no dehydration. Dehydration is recognized as an important, ever-present problem and instructions were issued requiring hydration either by mouth or parenterally at the earliest opportunity. Dehydration is not considered as great a danger, however, in winter as in summer. Some serum albumin was used successfully by the Marines during operations in the winter of 1950-1951.

Immediately following the Council's instructions in this matter, the Surgeon General of the Army directed that human serum albumin should be used by medical aid men in the field in subfreezing temperatures. In addition to pointing out the danger of dehydration, the directive made it clear that plasma would continue to be used in medical installations under cover where low temperatures posed no problem, and in both field and hospital facilities when temperatures were above freezing.

The Effect of Cortisone on Frostbite Injury[1]

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P. H. Dirstine, *Ph. D.*

FROSTBITE has assumed some prominence in recent phases of the Korean campaign as it has in other conflicts occurring in cold weather. The traumatic effect of cold is negligible under peacetime conditions when a person can take precautions or withdraw from the conditions causing injury. In that sense, frostbite is a preventable injury. Under conditions of war, however, several factors operate to increase the incidence of cold injury. Among these are inexperience of troops under cold weather conditions and failure to recognize the onset or implication of cold injury. Other factors adding to cold injury under war conditions are continued exposure with no opportunity to withdraw and the additional factor of dampness of clothing from perspiration or from contact with water.

Frostbite has come, through usage, to imply the cold injury which is incurred as the result of acute exposure of an extremity to temperatures below freezing. It is thus to be distinguished from general hypothermia (such as by immersion in cold sea water) and "immersion foot" (a term applied in World War II and implying the peripheral vascular neuropathy after chilling) which is due to prolonged exposure of the feet to moderate cold and wet conditions.

[1] From the Metabolic Research Facility, U. S. Naval Hospital, Oakland, Calif.

Frostbite injury is described as:

First degree when there is injury of the skin only, without blisters. Healing takes place without residual scar.

Second degree when the injury is localized to the skin, with the formation of blisters. Residual local hyperesthesia, and sensitivity to cold may occur.

Third degree when there is more severe damage, involving subcutaneous tissue, muscle, or bone. Total freezing of an extremity is rare. Sequelas depend on the extent of necrosis. Following the demarcation of viable tissue in 3 or 4 weeks, it is common to find more living tissue than was expected.

TABLE 1. *Comparison of cortisone-treated and control groups*

Patient	Age (years)	Duration of disease (days)	Extent of frostbite
Group 1 (control)			
1	22	24	Distal one-third of both feet; heels of both feet.
2	21	18	All toes.
3	20	22-25	All toes.
4	48	14	All toes of right foot.
5	20	25	All toes of left foot.
6	28	23	Distal segments of all toes.
7	23	20	All toes.
8	21	18	Great toes of both feet.
Group 2 (treated)			
9	19	22	All toes; sole of right foot.
10	20	22	All toes; soles and heels of both feet. All fingers.
11	20	26	Great toes of both feet; second toe of right foot.
12	20	22	All toes of right foot.
13	20	28	Tips of first 3 toes of both feet.
14	20	25	Fingers of right hand.
15	21	25	All toes of right foot; first and second toes of left foot.
16	19	19	All toes of both feet.

Much has been written concerning the pathogenesis of frostbite. Essentially, it is necrosis of more or less tissue following the anoxia induced by freezing. The extremities are involved most often because of their relative lack of insulation by fat tissue, their exposure, and, in the case of the feet, by the effect of water content in the foot covering because water conducts heat over 20 times more rapidly than air.

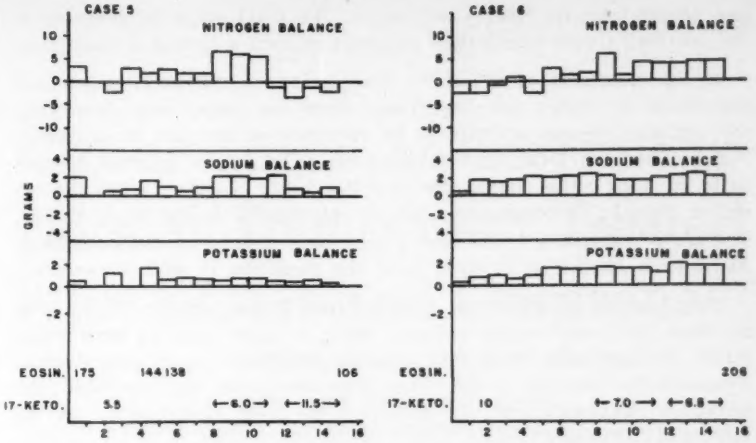

Figure 1. Studies of two control patients.

In the early stages of cold injury, when prevention of frostbite might be possible, there is little pain and sensations of cold and numbness are the only warning that freezing is occurring. These sensations are adequate warning to persons who are familiar with frostbite, but may be ignored by the inexperienced. The progress of the injury, if unchecked, is through the stage of vasoconstriction and anoxia, with blue-white or red skin. The part is stiff to the touch. As thawing takes place, a painful period of hyperemia is observed. The skin becomes intensely pink. Hyperemia continues with blister formation

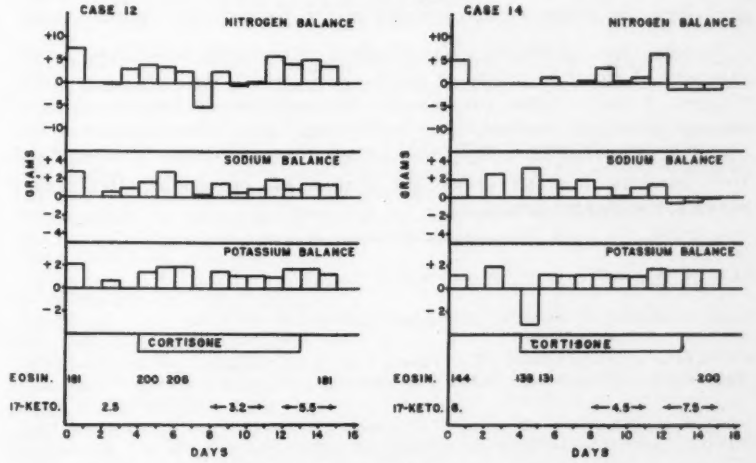

Figure 2. Studies of two cortisone-treated patients.

and severe pain for from 3 to 7 days. The final stage is gangrene of the involved tissue which then proceeds to form a line of demarcation.

In the treatment of frostbite, recognition of the early signs and prevention of injury are important. After the injury has developed, the optimum therapy appears to be exposure of the part in dry air at 70° F., with no local heating measures. The use of heparin appears to be of some value during the first few hours of injury when agglutinative thrombi theoretically might be prevented. In the later stages, Buerger's exercises and use of the part appear to be beneficial. Amputation should be delayed until the gangrene is well demarcated.

This hospital received 586 patients from Korea with frostbite, many of whom had complicating wounds. Many of these patients were transferred to hospitals near their homes. Inasmuch as no satisfactory treatment for frostbite in the stage after rewarming was available, the Metabolic Research Facility undertook to make observations on the effects of cortisone.

Material and methods. A project was set up to administer cortisone to a group of 8 patients and to use 8 other untreated patients as controls. All patients were placed on a standard diet with a fixed calculated intake of calories, nitrogen, sodium, potassium, and phosphorus. Balance studies were conducted on these elements. Antibiotics were used when indicated and physical activity about the ward was encouraged. The treated patients and controls were "paired" as to severity of injury insofar as possible. The treated group of patients received 100 mg. of cortisone in one intramuscular dose daily. Because no satisfactory placebo was found, none was used in the untreated group. Table 1 gives the ages of the patients, duration of injury, and describes the extent of involvement of the treated and control groups.

Results. No significant clinical effect of cortisone was noted on the progress of frostbite injury at the stage in which the agent was used. Figures 1 and 2 show the results obtained from balance studies on urinary nitrogen, sodium, and potassium; and observations on the circulating eosinophil count, and urinary 17-ketosteroids in representative patients in each group. No significant metabolic effects were noted in the treated group.

Observations on Essentially Normal Young Men Treated With ACTH and Cortisone[1, 2]

II. Effects on Tuberculin, Dick, and Schick Tests

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REPORTS on the effects of ACTH and cortisone on the tuberculin test disagree. Some investigators described inhibition in man (3-5), in tuberculous guinea pigs (6), and in hyperimmune guinea pigs (7, 8); others have noted no inhibition in tuberculous guinea pigs (9) and possibly increased severity of the reaction in tuberculous guinea pigs (10). During our study of the effects of ACTH and cortisone on the healing of frostbite lesions, an opportunity was afforded for studying the response of these subjects to tuberculin, Dick, and Schick tests.

(1) From the Cold Injury Section, Osaka Army Hospital.

(2) The second of three articles on this subject. The statistical analyses were made by H. M. C. Luykx, D. Sc.

(3) Thorn, G. W.; Forsham, P. H.; Frawley, T. F.; Hill, S. R., Jr.; Roche, M.; Staehelin, D.; and Wilson, D. L.: Clinical usefulness of ACTH and cortisone. New England J. Med. 242: 783-793, May 18, 1950.

(4) Long, J. B., and Favour, C. B.: Ability of ACTH and cortisone to alter delayed type bacterial hypersensitivity. Bull. Johns Hopkins Hosp. 87: 186-202, Sept. 1950.

(5) Fischel, E. E.: Relationship of adrenal cortical activity to immunity responses. Bull. New York Acad. Med. 26: 255-260, Apr. 1950.

(6) Derbes, V. J.; Dent, J. H.; Weaver, N. K.; Vaughan, D. D.: Response of tuberculin skin test to ACTH and cortisone in tuberculous guinea pigs. Proc. Soc. Exper. Biol. & Med. 75: 423-426, Nov. 1950.

(7) Osgood, C. K., and Favour, C. B.: Personal communication to Thorn, G. W. et al. See reference footnote (3).

(8) Stoerk, H. C.: Inhibition of tuberculin reaction by cortisone in vaccinated guinea pigs. Federation Proc. 9: 345, 1950.

(9) Sheldon, W. H.; Cummings, M. M.; and Evans, L. D.: Failure of ACTH or cortisone to suppress tuberculin skin reaction in tuberculous guinea pigs. Proc. Soc. Exper. Biol. & Med. 75: 616-618, Nov. 1950.

(10) Spain, D. M., and Molomut, N.: Effects of cortisone on development of tuberculous lesions in guinea pigs and on their modification by streptomycin therapy. Am. Rev. Tuberc. 62: 337-344. Oct. 1950.

TUBERCULIN TEST

Method. As previously described *(11)* the subjects were patients with frostbite lesions localized in the toes, fingers, or heels. The ACTH group consisted of 13 subjects, the cortisone group of 22 and the control group of 26. The distribution of white and Negro troops was random and, when analyzed separately, showed no significant difference in incidence and in reaction. There was no significant difference among the 3 groups relative to time in hospital or duration of hormone therapy prior to the skin tests, which were performed in all 3 groups on the same days.

Twenty mg. of ACTH was administered intravenously in one liter of normal saline solution or 5 percent dextrose in water daily for 31 consecutive days. The infusion lasted about 6 hours. Cortisone was administered orally as follows: 400 mg. (100 mg. every 6 hours) on the first day; 300 mg. (75 mg. every 6 hours) on the second day; and 200 mg. daily (50 mg. every 6 hours) until the twelfth day, at which time 200 mg. (100 mg. at 0800 and at 2000) was given daily through the thirty-first day.

TABLE 1. *Tuberculin test on twenty-fifth day of therapy*

Group	Number	Negative	Positive		1 plus	2 plus	3 plus	4 plus
			Number	Percent				
ACTH	13	4	9	69	2	4	2	1
Cortisone	22	7	15	68	2	3	8	2
Control	26	9	17	65	1	4	11	1

Second strength tuberculin (0.005 mg. PPD) in 0.1 ml. was injected intradermally in the right forearm. The reaction was measured at 48 hours and classified as follows:

Negative - Induration and erythema less than 5 mm. in diameter.
Positive - One-plus induration and erythema 5 to 10 mm. in diameter.
Two-plus induration and erythema 11 to 20 mm. in diameter.
Three-plus induration and erythema over 20 mm. in diameter.
Four-plus induration, erythema and necrosis; lesion over 20 mm. in diameter.

(11) Appel, S. B., and Orr, K. D.: Observations on essentially normal young men treated with ACTH and cortisone. I. Effects on glucose metabolism. U. S. Armed Forces M. J. 3: 207-220. Feb. 1952.

Results. Table 1 shows the results of the tuberculin tests on the twenty-fifth day of therapy as measured at 48 hours. There was no significant difference between the combined ACTH and cortisone groups compared with the control group (the critical ratio was 0.26; P was 0.8). Roentgenograms of the chest of all the subjects were normal. There was no past personal history of tuberculosis in any subject. Two control subjects had tuberculous mothers, and both of them exhibited 3-plus reactions. Because of the frequency of strongly positive, slowly resorbing reactions in all groups, a second test was performed on the twenty-first day after cessation of therapy only in those subjects with previously negative reactions. In the ACTH group 3 were negative and 1 was one plus. In the cortisone group 5 were negative, 1 was one plus, and 1 was two plus. In the control group 4 were negative and 1 was one plus. This degree of change is not significant *(12, 13)*.

DICK TEST

Method. The subjects were the same as described previously except for variations mentioned below. One-tenth milliliter of diluted scarlet fever streptococcus toxin was injected intradermally in the right forearm. A control solution was prepared by heating a vial of the same preparation at about 96° C. for 45 minutes *(14)*, and 0.1 ml. was injected intradermally in the left forearm. The reactions were observed and measured between 20 and 24 hours after injection and classified as follows:

Negative: Erythema in test arm less than 10 mm. in any diameter.

Negative with pseudo-reaction: Erythema in test and control arm, both being greater than 10 mm. in any diameter and of about equal magnitude.

Positive: Erythema in test arm greater than 10 mm. with control arm erythema less than 10 mm.

Positive with pseudo-reaction: Erythema in both arms greater than 10 mm. but much larger in the test arm as compared to the control arm.

Results. In the ACTH group (table 2) 11 subjects were tested on the twelfth day of therapy and on the ninth day *after* therapy and 2 subjects were tested only once (on the twenty-third and twenty-fifth days of therapy respectively). On the ninth day *after* therapy (table 5) the three positive reactors were positive-pseudo, negative, and negative-pseudo. The other 8 subjects showed no change. No negative reactor

(12) Criep, L. H.; Levine, M. I.; and Aaron, T. H.: Inhibition of tuberculin type reaction by antihistaminic drugs and rutin. Am. Rev. Tuberc. 59: 701-706, June 1949.

(13) Ellison, R. T.: Clinical studies on allergy to tuberculin following primary tuberculous infection; clinical value of recognizing state of secondary negative allergy. Am. Rev. Tuberc. 58: 463-475, Oct. 1948.

(14) Topley, W. W. C., and Wilson, G. S.: Topley and Wilson's Principles of Bacteriology and Immunity. 3d edition (revised by G. S. Wilson and A. A. Miles). The Williams and Wilkins Co., Baltimore, Md., 1946. vol. II, p. 1467.

TABLE 2. *Dick test: ACTH group**

Subject	On 12th day of therapy			9 days after therapy		
	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation
1	12 by 12	5 by 5	Positive	15 by 15	10 by 10	Pos.-Pseudo**
2	12 by 12	3 by 3	Positive	9 by 8	9 by 7	Negative
3	0	0	Negative	0	0	Negative
4	0	0	Negative	4 by 3	0	Negative
5	13 by 13	12 by 12	Neg.-Pseudo	12 by 12	15 by 15	Neg.-Pseudo
6	11 by 11	0	Positive	12 by 11	14 by 12	Neg.-Pseudo
7	0	0	Negative	0	0	Negative
8	0	0	Negative	0	0	Negative
9	0	0	Negative	0	0	Negative
10	20 by 15	10 by 10	Pos.-Pseudo	23 by 20	15 by 14	Pos.-Pseudo
11	0	0	Negative	0	0	Negative**

*Two subjects not included here were tested once on the twenty-third and twenty-fifth days respectively with negative reaction.
**The eosinopenia after giving ACTH intravenously at the time of this test was not over 50 percent.

became positive. Three subjects responded to an intravenous infusion of ACTH on the tenth day *after* therapy with an eosinopenia of less than 50 percent *(11)*.

In the cortisone group (table 3) 20 subjects were tested at the same time intervals described above. Because 12 subjects exhibited an eosinopenia of less than 50 percent at the time of the second test after an infusion of ACTH *(11)* the Dick test was repeated 1 week later. At this time only 6 subjects showed an eosinopenia of less than 50 percent. Of 2 initially positive reactors, 1 became negative-pseudo and of 15 negative reactors, 1 became positive and 2 negative-pseudo (table 5).

In the control group (table 4) 23 subjects were tested twice; 18 other subjects were chosen at random from available patients with similar mild frostbite lesions and were tested once. Two had a frankly positive reaction; 4 had positive-pseudo reactions; 33 negative; and 5 negative-pseudo reactions. Of 21 negative reactors retested 1 became positive; 1 positive-pseudo; and 1 negative-pseudo. If the positive and positive-pseudo reactions are classified as positive and the negative and negative-pseudo reactions as negative, no significant difference was observed between the ACTH and control group (the critical ratio was 1.6; P was 0.11). There was no significant difference between the combined ACTH and cortisone group compared with the control group (the critical ratio was 1.04; P was 0.32).

SCHICK TEST

Method. The subjects were those described previously. One-tenth milliliter of diluted diphtheria toxin stabilized with buffer was injected intradermally in the right forearm and 0.1 ml. of Schick test control solution (heat buffer toxin) was injected intradermally in the left forearm. Reactions were observed and measured at 3, 4, and 7 days. They were classified as in the Dick test. As in the tuberculin and Dick tests, the Schick tests were performed by the same investigators on the same day on all subjects and were interpreted by the same observers.

Results. In the ACTH group on the sixteenth day of therapy 11 subjects were Schick tested; 3 showed positive reaction (table 6). Ten days *after* therapy one initially positive reactor was negative. At this time 3 subjects had an eosinopenia of less than 50 percent after ACTH was given intravenously *(11)*.

In the cortisone group 20 subjects were tested on the sixteenth day of therapy and 10 days *after* therapy (table 7). At the time of the second test 12 subjects had an eosinopenia of less than 50 percent after ACTH was given intravenously *(11)*. Therefore, the entire group was retested 20 days after discontinuing the cortisone therapy. At the time of this test, 6 subjects had an eosinopenia of less than 50 percent. Subjects 15, 29, and 30 showed changes from positive to negative, negative to positive, and negative to negative-pseudo, respectively.

TABLE 3. *Dick test: cortisone group**

Subject	On 12th day of therapy			9 days after therapy			18 days after therapy		
	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation
12	0	0	Negative	0	0	Negative**	0	0	Negative**
13	13 by 12	12 by 10	Neg.-Pseudo	12 by 9	10 by 10	Neg.-Pseudo	15 by 12	15 by 10	Neg.-Pseudo**
14	11 by 8	8 by 8	Negative	16 by 12	0	Positive	15 by 15	10 by 10	Pos.-Pseudo
15	0	0	Negative	12 by 12	11 by 9	Neg.-Pseudo**	0	0	Negative
16	0	0	Negative	0	0	Negative	0	0	Negative
17	0	0	Negative	0	12 by 10	?**	0	0	Negative**
18	0	0	Negative	0	10 by 8	?	0	0	Negative
19	0	0	Negative	0	0	Negative	0	0	Negative
20	0	0	Negative	0	0	Negative	0	0	Negative
21	0	0	Negative	11 by 9	0	Positive**	0	0	Negative**
22	32 by 20	25 by 24	Neg.-Pseudo	30 by 30	15 by 10	Pos.-Pseudo	25 by 20	30 by 25	Neg.-Pseudo
23	15 by 15	8 by 8	Positive	15 by 14	6 by 6	Positive**	15 by 14	8 by 8	Positive
24	0	0	Negative	11 by 10	3 by 2	Positive**	0	0	Negative
25	0	0	Negative	8 by 8	0	Negative**	15 by 11	5 by 5	Positive
26	22 by 18	13 by 10	Pos.-Pseudo	30 by 26	22 by 20	Pos.-Pseudo**	25 by 20	12 by 10	Pos.-Pseudo
27	0	0	Negative	8 by 5	0	Negative**	0	0	Negative**
28	0	0	Negative	0	0	Negative**	0	0	Negative**
29	0	0	Negative	15 by 15	15 by 15	Neg.-Pseudo**	15 by 12	10 by 7	Neg.-Pseudo
30	15 by 15	6 by 6	Positive	18 by 15	18 by 15	Neg.-Pseudo**	12 by 11	12 by 10	Neg.-Pseudo
31	0	0	Negative	6 by 4	0	Negative**	0	0	Negative**

*Two subjects not included here were tested once on the twenty-seventh and twenty-eighth day of therapy with following reactions respectively: 30 by 28 and 34 by 26 (neg.-pseudo); and 8 by 8 and 2 by 2 (neg.).

**Eosinopenia after giving ACTH intravenously at the time of this test was not over 50 percent.

TABLE 4. *Dick test: control group*

Subject	On 12th day of therapy			9 days after therapy		
	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation
32	0	0	Negative	0	0	Negative
33	—	—	—	11 by 10	0	Positive
34-38	0	0	Negative	0	0	Negative
39	0	0	Negative	0	6 by 6	Negative
40	50 by 30	17 by 14	Pos.-Pseudo	15 by 15	10 by 10	Pos.-Pseudo
41, 42	0	0	Negative	0	0	Negative
43	6 by 6	8 by 8	Negative	20 by 12	12 by 11	Pos.-Pseudo
44	0	0	Negative	0	0	Negative
45	0	0	Negative	3 by 3	0	Negative
46	0	0	Negative	0	0	Negative
47	20 by 18	18 by 15	Neg.-Pseudo	22 by 15	20 by 20	Neg.-Pseudo
48-50	0	0	Negative	0	0	Negative
51	0	0	Negative	10 by 8	2 by 1	Positive
52	0	0	Negative	12 by 10	10 by 10	Neg.-Pseudo
53-55	0	0	Negative	0	0	Negative
61	—	—	—	0	0	Negative
62	—	—	—	15 by 15	15 by 12	Neg.-Pseudo
63-66	—	—	—	0	0	Negative
67	—	—	—	20 by 17	10 by 10	Pos.-Pseudo
68	—	—	—	10 by 10	10 by 8	Neg.-Pseudo
69-73	—	—	—	0	0	Negative
74	—	—	—	25 by 20	18 by 15	Pos.-Pseudo
75	—	—	—	20 by 20	15 by 13	Pos.-Pseudo
76, 77	—	—	—	0	0	Negative

TABLE 5. *Summary of results of Dick test*

ACTH group	During therapy		9 days after therapy			
	Interpretation	Number	Positive	Positive Pseudo	Negative	Negative Pseudo
	Positive	3	–	1	1	1
	Pos.-Pseudo	1	–	1	–	–
	Negative	8*	–	–	6	–
	Neg.-Pseudo	1	–	–	–	1
	Total	13	–	2	7	2
Cortisone group			18 days after therapy			
	Positive	2	1	–	–	1
	Pos.-Pseudo	1	–	1	–	–
	Negative	15	1	1	12**	1
	Neg.-Pseudo	2	–	–	–	2
	Total	20	2	2	12	4
Control group***			9 days after therapy			
	Positive	0	–	–	–	–
	Pos.-Pseudo	1	–	1	–	–
	Negative	21	1	2	18	–
	Neg.-Pseudo	1	–	–	–	1
	Total	23	1	3	18	1

*Two subjects were tested only once (i. e. during therapy).
**Six subjects did not demonstrate eosinopenia of over 50 percent after ACTH was given intravenously at the time of this test.
***Eighteen subjects were tested only once.

In the control group 24 subjects were tested twice, and 16 additional patients with similar mild frostbite lesions selected at random were tested once (table 8). In 2 the reaction changed from negative to positive-pseudo, and in 3 from positive to negative. The results of the Schick test are summarized in table 9.

Statistical examination of the Schick test results indicates: (1) no significant difference in the initial tests comparing the ACTH group alone with the control group (the critical ratio was 0.57; P was 0.40); (2) no significant difference in the initial tests when the hormone groups were combined and compared with the control group (the critical ratio was 1.6; P was 0.11); (3) no significant difference in the frequency of

changes in reaction in the ACTH as compared with the control group (the critical ratio was 0.92; P was 0.37); and (4) no significant difference in the frequency of changes in reaction of the combined hormone groups as compared with the control group (the critical ratio was 1.75; P was 0.07).

TABLE 6 *Schick test: ACTH group*

Subject	On 16th day of therapy			10 days after therapy		
	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation
1	12 by 10	0	Positive	14 by 13	0	Positive*
2-6	0	0	Negative	0	0	Negative
7	0	0	Negative	0	0	Negative*
8	0	0	Negative	0	0	Negative
9	10 by 10	0	Positive	12 by 11	0	Positive
10	14 by 14	0	Positive	0	0	Negative
11	0	0	Negative	0	0	Negative*

*Eosinopenia after giving ACTH intravenously at the time of this test was not over 50 percent.

DISCUSSION

That satisfactory adrenocortical stimulation was achieved is evident in the studies of eosinopenia following both ACTH and cortisone *(11)*. As reported elsewhere *(11, 15)*, the ACTH group exhibited side-effects consistent with hyperadrenocorticalism, although the time of actual stimulation provided by the intravenous technic of ACTH administration was probably not greater than 8 hours daily *(11)*.

The reported incidence of a positive tuberculin reaction in man has varied widely among observers mainly because of differences in environment (rural versus urban) and age of the subjects studied *(16)*. The remarkable similarity in reaction in the subject groups of this paper indicates that from 65 to 70 percent of troops show a positive reaction to the second strength tuberculin test.

The tuberculin type reaction, involving the neutralization of endotoxins *(17)*, represents a special class of hypersensitivity reaction inasmuch as the tissue cells themselves seem to be the site of the

(15) Appel, S. B.; Fulton, L. A.; and Orr, K. D.: Observations on essentially normal young men treated with ACTH and cortisone. III. Side-effects during therapy. To be published.

(16) Sweany, H. C.: Tuberculin test; its use, limitations and future possibilities in diagnosis. Am. Rev. Tuberc. 56: 135-156, Aug. 1947.

(17) Ratner, B.: Allergy, Anaphylaxis and Immunotherapy; Basic Principles and Practice. The Williams and Wilkins Co., Baltimore, Md., 1943. p. 44.

TABLE 7. *Schick test; cortisone group**

Subject	On 16th day of therapy			10 days after therapy			20 days after therapy		
	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation
12-14	0	0	Negative	0	0	Negative	0	0	Negative
15	12 by 12	0	Positive	15 by 15	12 by 12	Neg.-Pseudo	0	0	Negative
16-18	0	0	Negative	0	0	Negative	0	0	Negative
19	0	0	Negative	5 by 5	0	Negative	0	0	Negative
20	0	0	Negative	0	0	Negative	0	0	Negative
21	0	0	Negative	8 by 8	0	Negative	0	0	Negative
22	0	0	Negative	0	0	Negative	0	0	Negative
23	10 by 10	0	Positive	0	0	Negative	11 by 10	8 by 8	Positive
24, 25	0	0	Negative	0	0	Negative	0	0	Negative
26	10 by 10	0	Positive	0	0	Negative	0	0	Negative
27, 28	0	0	Negative	0	0	Negative	0	0	Negative
29	0	0	Negative	10 by 8	8 by 8	Positive	—	—	—
30	0	0	Negative	6 by 6	6 by 4	Negative	15 by 10	12 by 10	Neg.-Pseudo
31	0	0	Negative	0	0	Negative	0	0	Negative

*On the tenth day after therapy an eosinopenia of over 50 percent after giving ACTH intravenously was *not* demonstrated by subjects 12, 15, 17, 21, 23-28, and 30, 31. On the seventeenth day after therapy such an eosinopenia was *not* demonstrated by subjects 12, 17, 21, 27, 28 and 31.

TABLE 8. *Schick test: control group*

Subject	On 16th day of therapy			10 days after therapy		
	Test (mm.)	Control (mm.)	Interpretation	Test (mm.)	Control (mm.)	Interpretation
32	0	0	Negative	0	0	Negative
33	12 by 11	0	Positive	10 by 8	8 by 6	Positive
34	0	0	Negative	20 by 15	10 by 10	Pos.-Pseudo
35	0	0	Negative	0	0	Negative
36	15 by 12	0	Positive	0	8 by 6	Negative
37	3 by 3	3 by 3	Negative	5 by 5	8 by 6	Negative
38	0	0	Negative	0	0	Negative
39	12 by 11	0	Positive	15 by 10	0	Positive
40	8 by 8	0	Negative	0	0	Negative
41	12 by 11	0	Positive	0	0	Negative
42, 43	0	0	Negative	0	0	Negative
44	0	0	Negative	8 by 8	4 by 4	Negative
45	15 by 15	0	Positive	14 by 12	0	Positive
46	15 by 14	0	Positive	13 by 12	0	Positive
47	10 by 10	0	Positive	0	0	Negative
48	25 by 20	0	Positive	16 by 15	0	Positive
49	0	0	Negative	0	0	Negative
50	25 by 18	0	Positive	26 by 22	0	Positive
51	0	0	Negative	0	0	Negative
52	0	0	Negative	6 by 6	6 by 5	Negative
53, 54	0	0	Negative	0	0	Negative
55	0	0	Negative	30 by 20	15 by 12	Pos.-Pseudo
68	–	–	–	0	6 by 8	Negative
69	–	–	–	30 by 25	0	Positive
70	–	–	–	14 by 12	0	Positive
71-73	–	–	–	0	0	Negative
74	–	–	–	14 by 8	0	Positive
75	–	–	–	0	0	Negative
76	–	–	–	10 by 8	0	Positive
77, 78	–	–	–	0	0	Negative
79	–	–	–	11 by 8	0	Positive
80-82	–	–	–	0	0	Negative
83	–	–	–	24 by 22	0	Positive

antigen and no humoral antibodies are found *(18)*. Studies were made of the Dick and Schick tests, involving the neutralization of exotoxins *(17)*, wherein the concentrations of humoral antibodies are invoked. The incidence of negative Dick tests in subjects of the age group observed here may be estimated as from 70 to 80 percent *(19)*. The incidence observed in this study (from 69 to 88 percent) appears somewhat higher

(18) Harvey, A. M.: Introduction to series of papers on studies on ACTH and cortisone. Bull. Johns Hopkins Hosp. 87: 349-353, Nov. 1950.

(19) Ratner, B.: See reference footnote *(17)*, p. 306.

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TABLE 9. *Summary of results of Schick tests*

ACTH group	During therapy		10 days after therapy			
	Interpretation	Number	Positive	Positive Pseudo	Negative	Negative Pseudo
	Positive Negative	3 8	2		1 8	
	Total	11	2		9	
Cortisone group			20 days after therapy			
	Positive Negative	3 17	1 1*		2 15**	1
	Total	20	2		17	1
Control group***			10 days after therapy			
	Positive Negative	9 15	6	2	3 13	
	Total	24	6	2	16	

*Subject 29 became positive 10 days after therapy and was not available for retesting.
**6 subjects did not show an eosinopenia of over 50 percent after ACTH was given intravenously at the time of this test.
***16 subjects were tested only once.

and may reflect the high incidence of beta-hemolytic streptococcal infections in the Far East. The incidence of negative reactions to the Schick test was reported (in 1924) to be about 90 percent in adults *(20)*. The slightly lower incidence (from 63 to 85 percent) demonstrated by the present study may be a reflection of differences in Schick test toxin, the wide range of antitoxin concentration producing a negative reaction *(21)*, or the decline of diphtheria in the past 27 years.

CONCLUSION

In a controlled study of the effects of ACTH and cortisone on the tuberculin, Dick, and Schick tests in essentially normal young men no statistically significant differences were noted between the reactions during and after therapy. No alteration in the frequency of pseudo-reaction hypersensitivity to the Dick and Schick control solutions was observed.

(20) Park, W. D., and Zingher, A.: See reference footnote *(17)*, p. 171.

(21) Marshall, M. S.: Borderline Schick test. J. Pediat. 37: 620-625, Oct. 1950.

Treatment of Periodontal Disease

Julius N. Obin, *Captain, U. S. A. F. (DC)*

A SUCCESSFUL treatment plan for the cure of periodontal disease involves: (1) removal of the cause, (2) removal of the effects produced, and (3) increasing tissue resistance to prevent recurrence. Frequently, conservative therapy fails to accomplish these results. This is usually due to lack of recognition and hence failure to remove all the causative factors. Because periodontal disease is the result of a combination of causes rather than any single cause, a thorough examination and complete diagnosis are essential. This should include a full series of radiographs and bite-wings, study models and an evaluation of all phases of the case. Some of the more common causative factors are occlusal trauma, poor oral hygiene, deficient nutrition, emotional tension, and systemic disease (table 1).

TABLE 1. *Systemic disease which may cause periodontal disease*

Faulty nutrition.

Debilitating diseases which lower body resistances:
- Syphilis
- Nephritis
- Tuberculosis
- Gastrointestinal disorders

Blood dyscrasias:
- Anemia
- Purpura
- Polycythemia
- Leukemia
- Granulocytopenia

Endocrine dysfunction:
- Pituitary disorders
- Diabetes mellitus
- Parathyroid disturbances
- Adrenal hypofunction

Occlusal trauma frequently can be corrected by the equilibration of the natural teeth (figs. 1 and 2). The aim of such a procedure is to achieve simultaneous contact between as many teeth as possible, not only in centric occlusion, but also in the protrusive position and

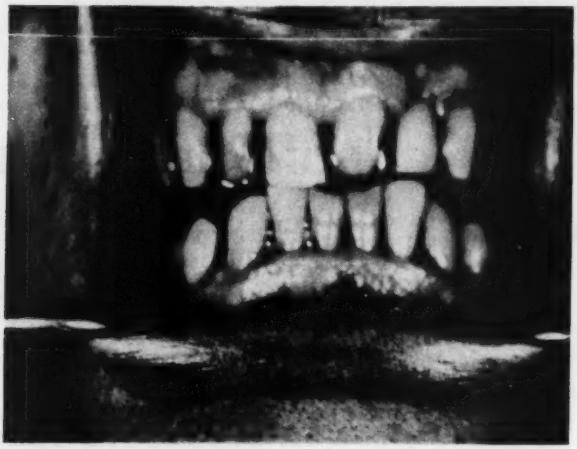

Figure 1. Traumatic relation in protrusive position.

protrusive and lateral excursions. This correction can also be achieved to a great extent by orthodontic technics and when possible this is the treatment of choice. Proper restorative dentistry is another method of reducing occlusal trauma. Combinations of these technics may be used.

Poor oral hygiene. Proper massage (preferably the modified Stillman method or, if the interproximate tissues are already destroyed, the Charters method) after the removal of irritating factors is the most frequently overlooked factor in the successful treatment of periodontal

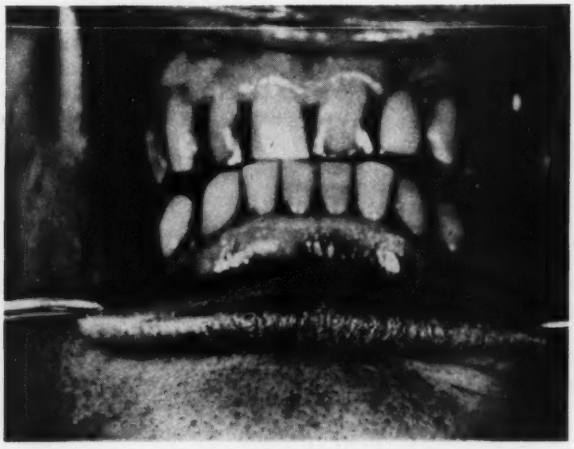

Figure 2. Correction of protrusive trauma. Note increased number of teeth in contact.

disease. Time should be expended in the teaching of gingival massage. A patient who is taught to massage effectively will rarely have a recurrence of gingival disease.

Nutrition. In addition to the minerals and vitamins in the foods, their detergent nature must be considered. Diets which emphasize refined carbohydrates as well as soft nonstimulating foods, lead to caries, poor gingival tone, and calculus deposits. Such foods do little to stimulate gingival circulation and functional hygiene. Unfortunately these are common fare with most patients. Vitamin supplements should be prescribed not only for their effect on gingival health but also as a means of improving gingival capillary tone and tissue texture.

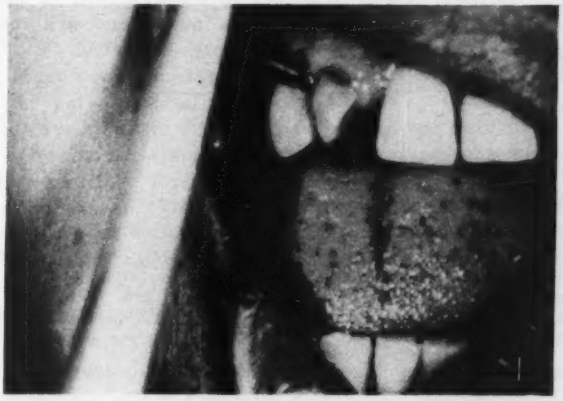

Figure 3. Use of interproximal stimulator.

The most frequently seen effects of periodontal disease are: ulatrophy (gingival recession), gingivitis, alveoloclasia, and pericementoclasia (pocket formation).

Too frequently, attempts are made to remove the symptoms rather than the cause. This may give temporary relief but the condition will usually recur. The use of penicillin to eliminate the signs of acute necrotizing ulcerative gingivitis is an example of treating symptoms without attempting to treat the cause of the disease. Another common method of treating necrotizing ulcerative gingivitis is the use of chromic acid solutions. The application of this severe caustic material cannot be recommended because of the resulting tissue destruction.

Gingivectomy without any other treatment should be discouraged. In most cases, pocket elimination is temporary because the underlying conditions creating the pockets, if not removed, will permit the formation of new areas of infection. In selected patients gingivectomy may be indicated but its routine use frequently results in an unesthetic appearance.

This building up of tissue resistance to prevent recurrence of gingival disease is often neglected. The patient should be encouraged in the proper and frequent use of the toothbrush and interproximal stimulator (fig. 3) because the dramatic results which follow conscientious application well repay the effort expended in its teaching and practice.

Steps in the treatment of periodontal disease, after a diagnosis based on a complete examination has been made, should consist of: (1) scaling, (2) instruction in massage. (3) dietary advice, (4) occlusal equilibration, (5) subgingival curettage, (6) polishing with a portepolisher, and (7) surgical procedures where indicated.

Adherence to these basic principles in the treatment of periodontal disease will give results of a permanent nature and will result in a lasting cure.

BOOK REVIEW

Prosthetic Dentistry, A Clinical Outline, by *F. Winston Craddock,* B. A., Dip. Ed., Cert. Dent. (N. Z.), M. S. D. (Northwestern), Head of the Department of Prosthetic Dentistry, University of Otago Dental School, Dunedin, New Zealand. 2d edition revised and enlarged. 363 pages; 179 illustrated. The C. V. Mosby Co., St. Louis, Mo., publisher, 1951. Price $6.75.

The second edition of this outline of prosthetic dentistry is a compactly written text that effectively reviews the fundamental principles of full and partial denture construction. In the revision of the preceding edition, which was printed in 1945, a new section on surgical preparation of the mouth from the prosthodontists's viewpoint is included and there is a discussion of splints that should be of interest to the dentist in the military service. Throughout the text the author makes wide use of the large bibliography of standard sources which he lists at the end of the book. The introduction to each subject is accompanied by a clear definition of terms which are often a source of controversy because of the varied meanings given them by different writers. The discussion on full denture construction in general follows the procedures most widely accepted today without attempting to stress any particular technic and suggests variations according to the nature of the individual problem. Although the discussion of partial dentures is not as extensive, the information on clasp function and load distribution is helpful. The author recognizes the technical skill achieved by the profession in dental prosthesis and emphasizes the need for an adequate recognition of the complexities of the prosthetic problem from an anatomic and physiologic point of view. This book should be as useful to the practitioner as it is to the student.—*Lt. Col. B. W. Dunn, U. S. A. F. (DC)*

Effects of Mobilization on 1950-1951 Residents and Interns at Approved Nonmilitary Hospitals as of 15 February 1951[1]

WHEN hostilities broke out in Korea, the manpower pool of physicians was estimated at 203,000. Of this number, 7,600 were on active military duty (2); another 32,000 reservists were potentially available for active military duty; and more than 163,000 or about 4 of every 5, were not liable for military service and neither actively nor inactively a part of the military medical corps. This distribution soon changed. Within a year, the number on active duty almost doubled, rising from 7,600 to 13,700 in June 1951. In October 1950 the 81st Congress enacted Public Law 779, the "doctor-draft" law, and the pre-Korea total of 32,000 physicians potentially available for military duty more than tripled; the number not liable for military service shrank from 163,000 to less than 77,000.

Physicians on active duty. About 6,000 physicians began their 24-month tour of duty in the Department of Defense during the first year of the Korean campaign. Three of every 10 were registrants under Public Law 779; the others were recalled reservists. About half of the registrants and reservists were in priority I and the balance were in priorities II, III, or IV, with most of them from priority IV, physicians with more than 21 months' service since 1941. Physicians potentially available for military service increased from 32,000 to a total of more than 114,000. The latter total included those of the 32,000 reservists not yet recalled to active duty (about 28,000) and about 86,500 registrants. The remaining physicians, men over 50 years of age, women, and aliens, were not liable for military service.

Registrants total over 88,000. About 88,400 physicians have registered in 1 of the 4 priority groups set up by Public Law 779. June figures from Selective Service showed 10,549 in priority I, about 2,500 in priority

(1) Condensed from a report prepared for the Health Resources Advisory Committee by the Health Resources Staff, Office of Defense Mobilization.

(2) In this report, physicians in the commissioned corps of the Public Health Service are considered as on active military duty.

III, over 30,300 in priority II, and more than 45,000 in priority IV. On active duty, as of 30 June, were 1,818, or 17 percent of those in priority I and 185, or 7 percent, of those in priority II.

No immediate need for priority IV physicians. Priorities I and II, the first groups to see military service, account for about 13,000 physicians. Since registration, this total has been reduced by about 2,000 who have entered military service. Another 2,500 will probably never serve because they are physically unfit, sole surviving sons, or have a deferrable dependency status. This leaves about 8,500 men remaining in priorities I and II who are potentially available. This group should be sufficient to provide replacements for the Army, Navy, and Air Force, at least through 1952 unless the size of the Armed Forces is sharply increased.

Men in priority III will probably not be called until sometime after January 1953. Because men in priority III will be called according to age, new graduates will form the bulk of those entering active military service when the supply of men in the first two priorities is exhausted. With each year the number of priority III men in each graduating class will continue to increase and by 1956 about 5,000 new priority III men will be added annually. Thus, those who originally registered as priority III men in January will probably not be called in the next 4 or 5 years, under the present authorized military strength. Because priority III will increase each year and there are at present more than 30,000 in this group, it is reasonable to assume that priority IV registrants will not be needed if present mobilization levels are continued. (These estimated call-up schedules do not apply to the estimated 1,000 reserve officers in priority III nor to those in priority IV.)

Residents and interns. About 24,500 of the 27,000 medical house staff members in training at the start of the 1950-1951 training year were in approved civilian hospitals (3). The remaining 2,500 were in the Department of Defense, 1,733; the Public Health Service, 269; and at unapproved civilian hospitals, 500. By mid-February 1951, training programs of each of these categories, with the exception of the Public Health Service, were reduced in varying degrees because of military withdrawals. For the country as a whole, the military mobilization of physicians had cut back postgraduate training for physicians by 9.5 percent. At approved civilian hospitals the loss was 7.8 percent. Hospitals of the Department of Defense reported the biggest reduction, a 37 percent curtailment, as their residents were reassigned from training to active duty. Less seriously affected were unapproved civilian hospitals (3 percent). Except in isolated cases, the hardship of military withdrawals due to Public Law 779 was not severe, chiefly because of the job done by the local advisory committees and the cooperation and understanding on the part of the Selective Service System and the Department of Defense.

(3) As used in this report, the term approved civilian hospitals includes those of the Veterans Administration and all nonfederal including nonprofit, proprietary, and those operated by city, county, and state governments.

The Artificial Kidney[1]

Paul E. Teschan, *Captain, MC, U. S. A.*

Marion E. McDowell, *Major, MC, U. S. A.*

ESTABLISHMENT of an artificial kidney at the Walter Reed Army Medical Center, under the joint auspices of the Army Medical Service Graduate School and the Walter Reed Army Hospital, has prompted a description which would acquaint medical officers of the Armed Forces with its practical therapeutic value and with its availability to patients of all Services at this center.

The artificial kidney, now in operation here, was developed in its essentials by Kolff (2) and subsequently modified in important respects by the group under the direction of Merrill et al. (3, 4). It is a simple device which succeeds in providing a membrane of large surface area between the patient's blood and the dialysate bath, across which diffusion of ions as well as small-molecule nonelectrolytes (for example, glucose, urea, and creatinine) may take place. Exposing blood to this diffusion process is termed *hemodialysis*. The circuit which carries blood at the rate of from 200 to 300 ml. per minute is shown in figure 1. Blood is led through disposable plastic tubes from the cannulated radial artery through a special rotating coupling to the surface of the revolving drum. A 120-foot cellophane tube (the diffusion membrane), wound in a continuous helix around the drum, carries a thin film of blood to its distal end whence it is led through another rotating coupling to a pump. This device brings the blood into a burette which also serves to remove air bubbles; from here it flows by gravity through a clot strainer into the patient's cannulated antecubital vein.

The dialysate bath (100 liters of water at 100° F.) is constituted by the addition of weighed amounts of certain normal extracellular fluid components. Table 1 lists its usual composition which may be altered to meet the needs of an individual patient such as one with potassium

(1) From the Army Medical Service Graduate School and the Walter Reed Army Hospital, Washington, D. C.

(2) Kolff, W. J.: New Ways of Treating Uraemia: The Artificial Kidney, Peritoneal Lavage, Intestinal Lavage. J. & A. Churchill, Ltd., London, 1947.

(3) Merrill, J. P.; Thorn, G. W.; Walter, C. W.; Callahan, E. J., III; and Smith, L. H., Jr.: Use of artificial kidney. I. Technique. J. Clin. Investigation 29: 412-424, Apr. 1950.

(4) Merrill, J. P.; Smith, S., III; Callahan, E. J., III; and Thorn, G. W.: Use of artificial kidney. II. Clinical experience. J. Clin. Investigation 29: 425-438, Apr. 1950.

intoxication. The diffusion process naturally reduces the concentration gradients between the normal concentrations of the dialysate bath constituents and their abnormal concentrations in the patient's plasma. Hence, the bath is emptied and reconstituted at suitable intervals during the procedure.

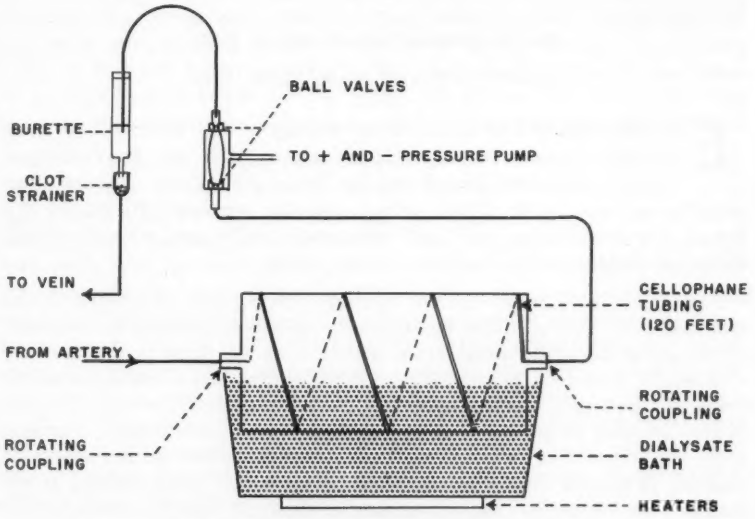

Figure 1. Circuit diagram of artificial kidney.

Heparin in the amount of about 150 mg. in 6 hours is given to prevent blood coagulation during the dialysis. Protamine sulfate is given intravenously at the conclusion to restore the blood coagulation time to normal. Dialysis is usually carried on for 6 hours. The blood pressure, pulse, and respiratory rate are taken every 15 minutes and recorded graphically by the nurse in attendance, who also administers regular nursing care as the dialysis proceeds.

USING THE ARTIFICIAL KIDNEY: ACUTE RENAL INSUFFICIENCY

Acute renal insufficiency as manifested by anuria or severe oliguria and the symptoms of acute uremia occur in a wide variety of circumstances, some of obvious military significance, and all of clinical importance. Military personnel, including battle casualties, may develop oliguria or anuria following (1) burns, (2) crush injuries, (3) sulfonamide administration, (4) mercuric chloride and carbon tetrachloride poisoning, (5) transfusion reactions, (6) prolonged, severe hypotension, (7) acute glomerulonephritis, (8) ureteral instrumentation, or (9) severe hepatic

failure (5). Often a combination of such causes occurs in these patients. Frequently the causative injury is sustained under the superimposed stress of battle. Acute prostatic obstruction, severe sodium chloride depletion, and hemorrhagic shock are causes of oliguria or anuria which may be reversed by urethral catheterization, sodium chloride administration, or whole blood transfusion and do not properly belong to the foregoing group in which renal tubular lesions of varying type and degree are uniformly present.

TABLE 1. *Dialysate bath*

Chemical constituents in 100 liters	Grams	Milliequivalents per liter of					
		Na	*K*	*Mg*	*Ca*	*Cl*	HCO_3
NaCl	660	113				113	
$NaHCO_3$	225	27					27
KCl	30		4			4	
$MgCl_2 \cdot 6H_2O$	22			2		2	
$CaCl_2 \cdot 2H_2O$	40				5	5	
Glucose	200						
Total		140	4	2	5	124	27
Normal plasma		142	5	3	5	103	27

These lesions correspond to the clinical oliguria or anuria which fails to respond with diuresis following the giving of fluids. Forcing fluids in an attempt to produce diuresis is regularly followed by peripheral and pulmonary edema and death in such patients.

The absence of functioning renal tissue accounts for the metabolic alterations of acute renal insufficiency. For practical purposes these may be grouped as follows: (1) azotemia, which reflects the failure of renal excretory function in disposing of metabolic end products; (2) metabolic acidosis, which reflects the failure of renal acid-base regulation; and (3) a tendency to potassium intoxication, which occurs in the presence of acidosis and azotemia, *may cause sudden death from cardiac standstill, and therefore is a medical emergency.*

POTASSIUM INTOXICATION

Anuria eliminates the major route of potassium excretion. Tissue catabolism, due to starvation and the stress of illness, liberates intracellular potassium to the extracellular fluid. The presence of acidosis

(5) Lucke, B.: Lower nephron nephrosis (renal lesions of crush syndrome, of burns, transfusions, and other conditions affecting lower segments of nephrons). Mil. Surgeon 99: 371-396, Nov. 1946.

enhances the tendency for potassium, normally present as the predominant intracellular cation, to move extracellularly (6, 7). When extracellular potassium concentration rises, progressive muscular weakness may be noted, but more significantly, the ECG reflects myocardial abnormalities by changes pathognomonic of potassium intoxication as shown in figures 2 and 3 (6). Once begun, this intoxication may progress to death in a few hours.

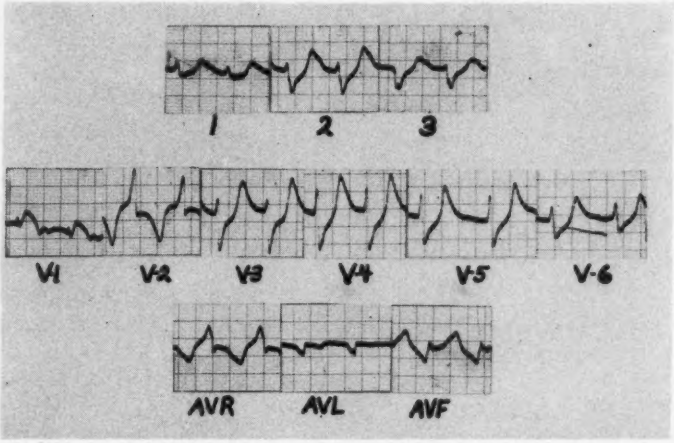

Figure 2. This tracing reflects severe potassium intoxication, manifested by absence of P waves, widened QRS complexes, steeply inclined ST segments, and high, peaked T waves. Chest leads V_2, V_3, and V_4 usually reveal these changes most strikingly.

The serum potassium concentration does *not* reflect the severity of the intoxication, that is, the imminence or remoteness of fatal cardiac standstill. For any elevated level of the serum potassium, the severity of the myocardial abnormality, as recorded by the ECG, is potentiated by low serum concentrations of sodium and calcium (6). Other factors may also be involved. The severity of the myocardial intoxication is shown by the ECG which, taken daily or every 12 hours in such patients, serves as a practical guide to the need for emergency therapy and/or for the artificial kidney. Fortunately, rapid intravenous infusion of from 200 to 400 ml. of 5 percent sodium chloride followed by an infusion of from 500 to 1,000 ml. of 15 percent dextrose with 1 unit of crystalline insulin per 2 grams of dextrose causes these changes to revert temporarily toward normal as the serum sodium concentration rises or as potassium is deposited intracellularly with glycogen (fig. 3). The chemical

(6) Merrill, J. P.; Levine, H. D.; Somerville, W.; and Smith, S., III: Clinical recognition and treatment of acute potassium intoxication. Ann. Int. Med. 33: 797-830, Oct. 1950.

(7) Elkinton, J. R.; Squires, R. D.; and Singer, R. B.: Intracellular cation exchanges in acidosis due to renal insufficiency; effects of alkali therapy. J. Clin. Investigation 30: 381-387, Apr. 1951.

abnormalities responsible for potassium intoxication tend to recur and at each recurrence become less susceptible to emergency therapy. Because hemodialysis produces a more lasting correction of these abnormalities, and in many patients represents the only practical means for removal of potassium, this intoxication is considered an absolute indication for use of the artificial kidney.

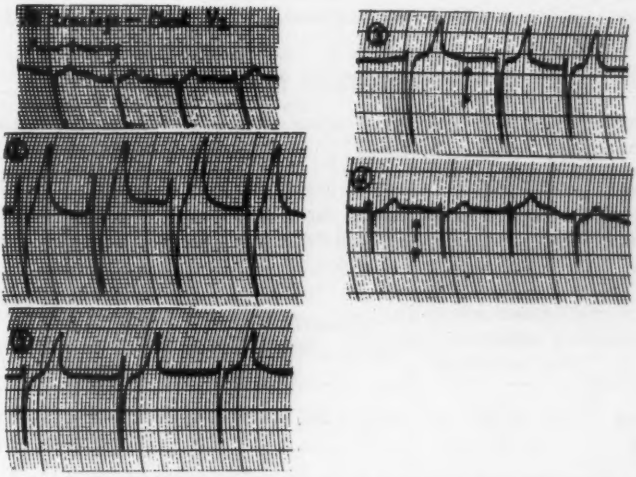

Figure 3. ECG of patient with potassium intoxication showing response to emergency treatment. Prior tracing was made on 14 March 1951 (blood pressure 148/68); tracing 1 was made at 0910 on 15 March (blood pressure 70/40); tracing 2 was made at 0930 after 100 ml. of 5 percent sodium chloride was given intravenously (blood pressure 100/55); tracing 3 was made at 1040 (blood pressure 130/70); and tracing 4 was made at 1830 after 200 ml. of 5 percent sodium chloride and 1.5 liters of dextrose-insulin solution was given intravenously (blood pressure 132/72).

The aims of therapy in acute renal insufficiency with oliguria should include: (1) the avoidance of overhydration by rigid control of fluid intake based on daily weights, estimated insensible water losses, and measured losses; (2) the suppression of tissue catabolism by administration of at least 100 grams of glucose daily *(8)*, and from 10 to 25 mg. of testosterone daily *(9)*; (3) the treatment of hypocalcemia and hyperphosphatemia with calcium and aluminum hydroxide gels *(10)*; (4) the treatment of clinical and severe chemical acidosis; and (5) prophylaxis

(8) Gamble, J. L.: Chemical Anatomy, Physiology and Pathology of Extracellular Fluid. 6th edition. Harvard University Press, Cambridge, Mass., 1947.

(9) Reifenstein, E. C., Jr., and Albright, F.: Metabolic effects of steroid hormones in osteoporosis. J. Clin. Investigation 26: 24-56, Jan. 1947.

(10) Shorr, E., and Carter, A. C.: Aluminum gels in management of renal phosphatic calculi. J. A. M. A. 144: 1549-1556, Dec. 30, 1950.

against fatal potassium intoxication by withholding potassium-containing fluids, suppressing tissue catabolism, and anticipating its sudden development.

In many patients conservative therapy alone will suffice and the patient may begin to form urine, frequently before the eleventh day. However, reversal of the chemical abnormalities by hemodialysis will frequently produce a marked clinical improvement, and transform a sick patient into an ambulatory patient who is not only more comfortable but able to eat.

RESULTS OF DIALYSIS

Several brief case summaries serve to illustrate the results that may be expected.

Case 1. A 23-year-old man had acute glomerulonephritis, with oliguria for 2 weeks and complete anuria for 5 days prior to dialysis. The ECG revealed marked potassium intoxication. The plasma concentrations before and after dialysis are shown below. Determinations on dialysate

	Blood urea nitrogen (mg. per 100 ml.)	*Creatinine* (mg. per 100 ml.)	Milliequivalents per liter of			
			Na	*K*	CO_2	*Cl*
Before	233	40	135	8.0	7.5	97
After	60	18	142	4.2	12.3	105

baths demonstrated the total removal of 93.7 grams of urea nitrogen, 127.4 grams of nonprotein nitrogen, and 380 milliequivalents of potassium. Within 24 hours after hemodialysis the patient had improved dramatically, was able to be out of bed and requested food which he tolerated well. During the following 9 days of continuing complete anuria, uremic symptoms gradually recurred, electrocardiographic signs of potassium intoxication reappeared and the patient was again dialyzed. The plasma concentrations before and after dialysis are shown below. The dialysate

	Blood urea nitrogen (mg. per 100 ml.)	Milliequivalents per liter of			
		Na	*K*	CO_2	*Cl*
Before	230	135	6.3	19.1	106
After	70	146	4.1	20.2	109

showed the removal of 64.2 grams of urea nitrogen and 400 mEq. of potassium. Again a marked clinical improvement occurred. The patient remained anuric for 14 days following the second dialysis. He died suddenly as a third dialysis was being arranged. Autopsy revealed (1) bronchopneumonia of the middle and lower lobes of the right lung, (2) massive pulmonary edema, and (3) kidneys of normal size with the

granular surface and punctate hemorrhagic areas of acute glomerulonephritis *(11)*.

Case 2 (12). A 71-year-old man with intravascular hemolysis produced by distilled water used as a bladder-irrigation fluid in a transurethral prostatectomy developed a hemoglobinuric nephrosis with oliguria 8 days prior to dialysis. The plasma concentrations before and after dialysis are shown below. The patient's good clinical response to dialysis was followed by a diuresis and ultimate recovery.

	Blood urea nitrogen (mg. per 100 ml.)	Milliequivalents per liter of			
		Na	*K*	CO_2	*Cl*
Before	179	128	5.9	10.3	89
After	50	140	4.6	15.5	111

Case 3 (12). A 35-year-old woman had preeclampsia with premature separation of the placenta. This patient was oliguric. For 5 days before dialysis she passed less than 100 ml. of urine daily. The ECG showed evidence of potassium intoxication. The plasma concentrations before and after dialysis are shown below. This patient had a postdialysis diuresis with uneventful recovery.

	Blood urea nitrogen (mg. per 100 ml.)	Milliequivalents per liter of			
		Na	*K*	CO_2	*Cl*
Before	137	113	9.5	11.8	83
After	63	129	5.0	18.2	104

OTHER INDICATIONS FOR DIALYSIS

Barbiturate intoxication. Patients who have ingested large doses, especially of the longer-acting barbiturates, become comatose and may develop hypotension and/or anuria. Renal clearance of barbiturates is poor. The renal tubules may reabsorb as much as 96 percent of the substance filtered at the glomeruli *(13)*. Hemodialysis provides a means of removing large amounts of barbiturate from such patients, thus avoiding the hazards of prolonged coma.

Salicylism. Overdoses of salicylates represent another type of intoxication by a dialyzable substance in which striking benefit may be expected from hemodialysis *(14)*.

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(11) Microscopic examination not completed when this article was in preparation.

(12) Abstracted from medical records of Peter Bent Brigham Hospital, Boston, Mass., courtesy of Dr. J. P. Merrill.

(13) Swan, Dr. R. C.: Personal communication.

(14) Doolan, P. D.; Walsh, W. P.; Kyle, L. H.; and Wishinsky, H.: Acetylsalicylic acid intoxication. J. A. M. A. 146: 105-106, May 12, 1951.

The oliguria and/or hypotension of severe hepatic failure. Oliguria and anuria in patients with hepatic failure also results in azotemia, acidosis, and potassium intoxication. Hypotension in these patients may respond dramatically to hemodialysis when all other measures have failed. Merrill et al. *(4)* recounts such an experience. Advantage is taken of the rise in blood pressure often recorded after about 2 hours of dialysis in any patient, regardless of his initial pressure.

Hepatic coma without oliguria or hypotension. Whether dialysis is of value in this condition is a matter of interest and requires investigation. In the absence of contraindications, and if the patient fails to respond to ordinary therapy, dialysis may be of benefit.

Symptomatic exacerbations in chronic uremia. Dehydration from any cause deprives the chronic uremic patient of his only real means of symptomatic and chemical control, that is, his ability to excrete a large volume of urine. His inability to concentrate urine under these circumstances results in sharp increases in azotemia and an intensification of symptoms. The resultant vomiting increases the dehydration and electrolyte abnormality. In such instances hemodialysis may be expected to restore the patient to his prior status.

Surgery in chronic uremia. Whereas an indwelling urethral catheter may help restore renal function after prolonged prostatic obstruction, circumstances may indicate or require an operation before azotemia has regressed. The operative risk involved may be lessened with a low level of azotemia.

CONTRAINDICATIONS TO DIALYSIS

Only one absolute contraindication exists for hemodialysis and that is overt bleeding from any source. The total heparinization of each patient to prevent hemocoagulation in the dialyzer naturally will accentuate blood loss from any bleeding site. Although stools producing a 2 or 3 plus guaiac test do not contraindicate the procedure, they suggest an added risk. The studies of Merrill et al. *(3, 4)* indicate that such possible hazards as hemolysis, platelet lysis, or leukocyte destruction from trauma to these elements in traversing the dialyzer circuit, do not occur to any significant extent. Hematocrits determined at intervals fail to reveal hemochromogen in the supernatant plasma; and circulating leukocyte and platelet counts determined after dialysis show no significant changes from their predialysis levels.

SUMMARY

Use of the artificial kidney is indicated for the correction of severe chemical abnormalities in patients with acute uremia. The clinical as well as the chemical improvement achieved by its use is striking. Potassium intoxication is a medical emergency and an absolute indication for dialysis. Although use of the artificial kidney is ordinarily not to be

considered lifesaving, the risk of losing a patient through potassium intoxication or other effects of uremic toxemia may thus be avoided. Other indications for hemodialysis include barbiturate intoxication, salicylism, hepatic coma with or without hypotension or oliguria, symptomatic exacerbations in chronic uremia, and its use as a preoperative measure in chronic uremia. Overt hemorrhage from any source is a contraindication to the use of the artificial kidney.

BOOK REVIEWS

Nutrition and Climatic Stress, With Particular Reference to Man, by *H. H. Mitchell,* Professor of Animal Nutrition, University of Illinois, and *Marjorie Edman,* Research Assistant in Animal Nutrition in Charge of Literature Survey, University of Illinois. 234 pages. Charles C Thomas, Publisher, Springfield, Ill., 1951. Price $6.75.

This comprehensive review of the literature was originally prepared by the authors under a contract between the University of Illinois and the Quartermaster Food and Container Institute for the Armed Forces. The value of this publication lies in the fact that the literature reviewed consists not only of that published in scientific periodicals but also in reports, many of which are obscure and not generally available. For this reason the extensive bibliography alone is of value. The material reviewed is divided into 3 broad categories: diet in a cold environment, diet in a hot environment, and diet at altitude. These 3 forms of stress are discussed in relation to the physiologic effects of the stress, the effect of the stress on nutritional requirements and the effect of diet on tolerance to the stress. Both animal and human experiments in this field are reviewed.—*Lt. Col. C. J. Koebn, MSC, U. S. A.*

Physical Medicine and Rehabilitation for the Aged, by *Walter S. McClellan,* M. D., Medical Director, The Saratoga Spa, Saratoga Springs, New York; Associate Professor of Medicine, Albany Medical College, Albany, N. Y. American Lecture Series Publication No. 105, A Monograph in American Lectures in Physical Medicine. 81 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1951. Price $2.

This monograph on physical medicine has many shortcomings. The author attempts to cover the whole field of physical medicine and rehabilitation as related to the aged in 64 pages. As a result, no one phase of the subject is given sufficient space. For the specialist in physical medicine or for the physical therapist and occupational therapist, the book would be a mere primer. Doctor McClellan, an authority on spa therapy, presents the advantages of hydrotherapy in the treatment of degenerative disorders. Even this section, however, could be ex-

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panded to advantage. I do not believe that this book has materially contributed to the field of physical medicine and rehabilitation and that the subject matter has been covered more completely in previously published texts.—*Lt. Col. J. N. Schaeffer, U. S. A. F. (MC)*

Clinical Pathology of the Eye, A Practical Treatise of Histopathology, by *Bernard Samuels,* M. D., Emeritus Clinical Professor of Ophthalmology, Cornell University Medical College; Consulting Ophthalmologist, New York Hospital; Acting Advisory Surgeon and Consulting Pathologist, New York Eye and Ear Infirmary, and *Adalbert Fuchs,* M. D., E. O. Professor of Ophthalmology, University of Vienna; Consulting Ophthalmologist and Pathologist, New York Eye and Ear Infirmary. 420 pages; 418 illustrations, 191 in color. Paul B. Hoeber, Inc., New York, N. Y., publisher, 1952. Price $20.

Aided by adequate numbers of plates and figures, the authors present in clear, concise writing the varied and diverse ocular lesions. In the first chapter important basic facts of pathology are reviewed, showing how they apply to the eye. In succeeding chapters the separate portions of the eye are taken up individually and the histopathologic findings are presented. The clinical aspects of each lesion are correlated with the anatomic changes. This, I believe, is the most valuable feature of the book. As an example, the section on retinal exudates is divided into groupings as noted clinically with the ophthalmoscope and the possible pathologic changes which will give such a picture are given. The last several chapters deal with such subjects as myopia, tuberculosis, postoperative complications, and injuries. The many possible lesions associated with each are discussed in detail. The section on postoperative complications is very well done and to the point. I wish it were more lengthy. The last chapter describes ocular tumors. The 44 color plates are excellent. Some have appeared previously in Fuchs' "Diseases of the Fundus Oculi," but this does not lessen their value. All plates, as well as figures, are accurate drawings rather than photographs. A number of controversial points, such as the cause of sympathetic ophthalmia, are well handled.—*Commander D. A. Mills, MC, U. S. N.*

From A Doctor's Heart, by *Eugene F. Snyder,* M. D., with a foreword by *Paul Dudley White,* M. D. 251 pages; illustrated. Philosophical Library, New York, N. Y., publishers, 1951. Price $3.75.

This book is the philosophic account of an episode of myocardial infarction suffered by the author, a physician. It describes the reactions of the patient, particularly those engendered by his physician, an eminent cardiologist, a psychiatrist friend, and a general practitioner. The author is a native of Russia who fled from the revolutionists to Czechoslovakia and later fled from the Nazis to this country. Much of the book is devoted to his viewpoint on these regimes and on the current international situation. The book is written for laymen and cannot be considered a scientific work.

—*Col. E. M. Goyette, MC, U. S. A.*

Nongonococcal Urethritis[1]

Robert S. Graham, *Lieutenant, junior grade, MC, U. S. N. R.* [2]

OVER a year ago, the Venereal Disease Control Section of the infirmary at Camp Lejeune, N. C. became concerned with the large number of cases of urethral discharge (not due to gonorrhea) in personnel at that station. Contrary to often expressed opinion, it soon became obvious that these were not cases of inadequately treated gonorrhea. The literature at hand had little to offer in the way of explanation as to the cause or therapy. Conferences with other medical officers in the Navy, as well as in the other military services, revealed this problem to be widespread both in the United States and in the Far East. Information from the Bureau of Medicine and Surgery indicated that some activities had reported up to 3 percent of their total personnel had been treated for this condition in a single month. Some larger stations in the continental United States reported an incidence of this condition 3 times as high as that of gonorrhea. As this had become one of our most perplexing problems in therapy and little was known about methods of control, a study of all such patients (totaling over 1,500) seen at the infirmary was begun.

There are many causes for a urethral discharge other than gonorrhea. These were formerly classified under the general term, nonvenereal urethritis. From among these a condition which seems to be a definite entity clearly related to sexual exposure appears to be responsible for this increased incidence. It has a prolonged incubation period, is of long duration, is complicated by prostatitis, and responds to various therapeutic measures. The facts suggest the question: Are we dealing with a sixth venereal disease? It would appear that this disease became prevalent after penicillin came into general use. The relationship of penicillin and other antibiotics to this form of urethritis, if any, may well prove of interest.

DIAGNOSIS

In order to identify and diagnose this particular form of urethritis, which we refer to as nongonococcal urethritis for lack of a more specific

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(2) Formerly VD Control Officer, Camp Lejeune, N. C.

name, the other causes of urethral discharge with which it may be confused must be eliminated.

Acute gonococcal urethritis usually occurs from 3 to 5 days after intercourse with an infected person and is characterized by a burning sensation in the urethra on urination followed by a profuse purulent discharge. It is diagnosed by smear and culture and responds well to penicillin therapy.

Urethral and prostatic infestations with Trichomas vaginalis result in a burning sensation in the urethra and a mucopurulent or slightly blood-tinged discharge. It is diagnosed easily by identifying the parasite in suspensions of prostatic secretion, urinary sediment, and/or urethral discharge. This condition is prevalent enough to warrant examination for this parasite in all patients with urethral discharge not due to gonorrhea. A course of quinacrine should be given to such patients.

Traumatic urethritis produced by excessive sexual activity or "milking down" the urethra by those over-anxious persons who frequently check themselves for a urethral discharge. This discharge is usually sparse and may be seen as a "tear" on arising in the morning. It may be accompanied by urethral burning on urination. The discharge is frequently mucopurulent. This form of urethritis usually subsides in several days if the trauma to the urethra is discontinued. Smears and cultures are usually devoid of organisms. Although no therapy is indicated, these patients do need reassurance.

Chemical urethritis produced by intra-urethral prophylaxis is occasionally reported. This usually develops in from 12 to 24 hours and is characterized by a burning sensation and a slight discharge in which no organisms can be found. Such forms of urethritis clear fairly rapidly if the urethra is permitted to rest without further irritation.

Normal urethral mucus. Some men, stimulated by talks on venereal disease or by anxiety after promiscuous sexual exposure, closely inspect their sexual organ and discover for the first time the normal urethral mucus. In some it is more profuse than in others. These men frequently become alarmed and report to their medical officer in the belief that they have a venereal disease. In such cases, reassurance is all that is necessary in the way of therapy.

Alcoholic urethritis. A burning sensation in the urethra during urination and a slight urethral discharge is seen in some persons after alcoholic excesses. The discharge is free of organisms and clears spontaneously in from 24 to 48 hours provided no more alcohol is consumed and the patient gets sufficient rest.

Normal prostatic secretion. In some who have not had sexual relief for a long time a "spilling over" of normal prostatic secretion occurs, usually while straining at stool, or as a urethral discharge seen on arising in the morning. Laboratory examinations of the discharge reveal normal prostatic secretion. The only therapy needed is reassurance.

An unclean preputial sac may give rise to infection of the anterior urethra. Such infections are treated effectively with soaks plus penicillin given systemically. Circumcision should be recommended where necessary and the patient instructed in personal hygiene.

Very rarely, *foreign bodies in the urethra* are found as the cause of a discharge.

Urethral irritations are found to result in some patients from *vaginal secretions or sensitizing vaginal jellies or douches.* Frequently in such patients, the skin of the penis reveals evidence of the sensitization.

Partial urethral obstructions or anatomic defects behind which bacteria may lodge and act as foci of infection are found in some patients with recurrent urethral discharges occurring over long periods of time.

CHARACTERISTICS OF NONGONOCOCCAL URETHRITIS

At Camp Lejeune, excluding all of the foregoing categories, there were still about 150 cases per month of the entity described below or roughly 3 times the incidence of gonorrhea. All such patients were studied for at least 3 days before any form of therapy was instituted. On each of the days of study, a gram-stained smear of the discharge was made. A culture was grown on chocolate media under carbon dioxide tension. With many patients an additional blood agar culture was taken and grown aerobically. Several hundred of these patients were interviewed by myself and other trained interviewers used in venereal disease contact reporting. By means of these interviews the following characteristics of the disease were determined:

1. The first symptom of the disease is usually a burning sensation in the urethra during urination. This is followed shortly by a urethral discharge.

2. The discharge is usually mucopurulent or purulent and is seldom as profuse as the usual discharge seen in gonorrhea.

3. The incubation period is usually at least 14 days after intercourse and frequently longer; rarely is it found to be shorter.

4. The course may be prolonged. Some patients reported that they had had this disease for from 6 months to several years.

5. A history of sexual exposure was elicited in virtually 100 percent of the patients. Almost all had failed to use mechanical prophylaxis or prophylactic salve. From foreign areas, reports of this disease in those who had taken penicillin prophylactically by mouth reveal that that form of prophylaxis is ineffective in preventing nongonococcal urethritis, although it is from 90 to 100 percent effective in preventing gonorrhea when used shortly after exposure.

6. This disease may exist concurrently with gonorrhea and be diagnosed when the urethral discharge continues after adequate treatment

with penicillin has cured the gonorrhea, because this entity does not respond to penicillin. Care should be taken in the laboratory to make sure that gonorrhea is not being overlooked in smears and cultures taken after treatment.

7. A large number of these patients develop prostatitis. No evidence of orchitis, epididymitis, arthritis, or other complications was found.

No remarkable bacteriologic evidence of a causative organism was found consistently. The usual surface organisms including the diphtheroids were found. A large gram-positive coccus which could not be identified by means of present classification systems was isolated from many patients. There is no evidence as yet of an etiologic role for this organism. Further work on all possible etiologic agents including viruses is necessary.

TREATMENT

Response to therapy was interesting. Penicillin proved to be of no value and some actually developed the condition while receiving penicillin for some other disease. In 1,000 of the series, sulfadiazine was tried and found to be effective in 90 percent of the patients within from 3 to 5 days, when an initial dose of 4 grams followed by 1 gram every 4 hours was given. Smaller initial doses of this drug are ineffective. Very few reactions to the sulfadiazine were observed as each patient was carefully instructed as to the specific amount of water to consume. All were treated as out-patients. For others in the field with limited water intake or excessive fluid loss, chloramphenicol was used as the next drug of choice, although it is not quite as efficacious as sulfadiazine. A dosage of 500 mg. every 6 hours was given to these patients. Aureomycin in doses of 500 mg. every 6 hours gave poor results when compared with those following the use of sulfadiazine or chloramphenicol.

No therapeutic agent was found to be of value in treatment unless the following were strictly avoided by the patient for a period of 2 or 3 weeks during and after treatment: (1) "milking down" the urethra to expel any discharge present, (2) excessive fatigue, (3) sexual intercourse, and (4) alcoholic beverages. In a few resistant cases, sitz baths, prostatic massages, and bed rest were used effectively along with the drug therapy. Local irrigations are to be avoided as they tend to prolong the inflammation.

SUMMARY

Reports from military activities of all 3 of the Armed Forces indicate an increasingly prevalent form of sexually acquired urethritis not due to the gonococcus, among service personnel. Other causes of urethral discharge were ruled out in a series of patients with this condition. Penicillin was entirely useless in treating these patients. Sulfadiazine was effective in 90 percent of 1,000 patients in the series treated with

that drug but only when an initial dose of 4 grams followed by 1 gram every 4 hours was used. Chloramphenicol was used in other patients where limitation or excessive loss of fluid was expected in connection with duties in the field. Aureomycin was found to be less effective than chloramphenicol. *No* drug is effective in these patients unless "milking down" the urethra, excessive fatigue, sexual intercourse, and alcoholic beverages are avoided. Penicillin given prophylactically by mouth does not protect against this disease, and widespread substitution of this prophylactic agent for mechanical methods may have contributed to the increasing prevalence of nongonococcal urethritis where it is used. The etiology of this disease is unknown. If a specific etiologic agent can be identified, it will constitute the sixth venereal disease.

BOOK REVIEW

Observations on the General Effects of Injury in Man, With Special Reference to Wound Shock, by *R. T. Grant* and *E. B. Reeve*. Privy Council. Medical Research Council Special Report Series No. 277. 313 pages. His Majesty's Stationery Office, London, publisher, 1951. Price about $1.20.

In this special report the authors record their clinical observations on traumatic shock. The study includes casualties from the London air raids and the Italian battle front. The authors' particular contribution is in the sphere of clinical definition. They believed with others that much discussion on the subject of "wound shock" was vitiated by the fact that different writers were discussing several different conditions. In a thorough study of about 300 patients suffering from severe trauma they found that various signs following such trauma occurred in a number of distinct patterns. They paid most attention to hemorrhage as the major contributing factor to traumatic shock and, accordingly, considered transfusion to be the most valuable form of treatment. By correlating clinical observations with carefully controlled blood volume measurements they succeeded in evolving simple and reliable clinical methods of estimating the amount of lost blood and required transfusion. Worthwhile observations on disturbances of water and electrolyte balance that may follow abdominal injury are given and the dangers of bad anesthesia and the rough handling of casualties are thoughtfully described. The clinical system evolved by Dr. Grant in recognizing phases of shock and estimating the degree have proved most workable in his hands.

—*Lt. Col. F. L. Bauer, MC, U. S. A.*

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Chronic Segmental Atelectasis[1]

William R. Beisel, *Captain, MC, U. S. A.*

James O. Gillespie, *Brigadier General, MC, U. S. A.*

CHRONIC pulmonary atelectasis localized to individual segments forms a distinct clinical entity which is now being recognized with increasing frequency. The importance of tuberculous hilar lymphadenopathy and endobronchial tuberculosis in causing this condition is worthy of emphasis. In 1920 Eliasberg and Neuland (2) introduced the term "epituberculosis" to describe the sudden appearance of a large, fairly uniform roentgenographic density in the lung fields of children who were undergoing a primary tuberculous infection. These patients were characterized by apparent excellent health despite the ominous appearance of the roentgenographic lesion, and eventual clearing was usually spontaneous and complete. Recent writers (3-9) have discarded the term "epituberculosis," believing that its features are those of a lobar or segmental atelectasis caused by pressure from enlarged hilar nodes. The major experimental work on the pathologic

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(1) From the Letterman Army Hospital, San Francisco, Calif.

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physiology of atelectasis was published by Coryllos and Birnbaum *(10, 11)* in 1929. With the rapid development of thoracic surgery and the increasing knowledge concerning the detailed anatomy of the tracheobronchial tree, special roentgenographic views and technics have been developed *(12-21)* for the diagnosis and localization of atelectasis in individual pulmonary segments.

Relatively few physicians, when confronted with the problem of the cause of chronic segmental atelectasis are aware of the high incidence of positive findings of tubercle bacilli by culture and guinea pig in these patients.

This article deals with a study at this hospital in the past 3 years of 12 adult patients showing chronic segmental atelectasis. This diagnosis is limited to patients showing a prolonged atelectasis of 1 or more of the named bronchial segments; in each instance possible etiologic factors such as neoplasm, foreign body, postoperative atelectasis, or widespread pulmonary tuberculosis have been ruled out. In 5 of these patients, tubercle bacilli were isolated by culture or guinea pig inoculation and in the remainder certain evidence incriminated calcified hilar lymph nodes as the probable cause of bronchial obstruction.

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(21) Stivelman, B. P.: Atelectasis in pulmonary tuberculosis. J. A. M. A. 99: 1666-1670, Nov. 12, 1932.

ETIOLOGY

A tuberculous process in hilar lymph nodes is the major single cause of chronic segmental atelectasis. Bronchial obstruction may be caused by extrinsic pressure from enlarged tuberculous nodes or by penetration of the caseous process through the bronchial wall. Atelectasis may occur during a primary tuberculous infection or after the tuberculous hilar node has become calcified and apparently quiescent *(3)*. Doig *(13)* reported 14 patients with atelectatic bronchiectasis of the middle lobe of the right lung which apparently dated back to an attack of pneumonia, measles, or pertussis, although most of the patients had a preceding pulmonary tuberculosis or were close contacts of persons with open tuberculosis. In a review of 300 children with atelectasis (diagnosed roentgenographically) Brjum *(12)* reported that 154 were caused by tuberculosis, 69 by influenza, 47 by pertussis, and 30 by measles.

Tuberculosis was excluded as an etiologic factor in each of the 12 patients with atelectasis of the middle lobe of the right lung reported by Graham et al. *(14)*. In these patients, microscopic examination of the nodes producing atelectasis by bronchial compression revealed only chronic nonspecific lymphadenitis.

PATHOGENESIS

It has been conclusively proved that complete obstruction of a bronchus is the only cause of atelectasis *(22)*. Such factors as (1) respiratory muscle paralysis, (2) bronchomotor and vasomotor nervous reflexes, (3) interference with thoracic movements by pain or narcotics, (4) extrapulmonary infection, and (5) general exhaustion may each contribute to the development of pulmonary atelectasis, but obstruction of a bronchus must be complete before atelectasis actually occurs.

The incidence of segmental or lobar atelectasis during the course of childhood tuberculosis has been well documented *(4-9, 12, 23)*. Inflamed tuberculous hilar nodes may compress or kink a bronchus by simple, extrinsic, mechanical pressure, leading to narrowing of the bronchial lumen and plugging with trapped secretions. This mechanism is most common in children where the bronchi are softer and more easily compressed than in adults. Slight changes in the degree of extrinsic pressure or in the amount of retained secretions may cause the bronchial obstruction to be intermittent. For this reason atelectasis

(22) Coryllos, P. N.: Importance of atelectasis in pulmonary tuberculosis; its relation to fibrosis and to pathogenesis and healing of tuberculous cavities. Am. Rev. Tuberc. 28: 1-26, July 1933.

(23) Stoloff, E. G.: Acute massive atelectasis of lung, complicating fresh tuberculous primary complex. Am. J. Dis. Child. 35: 239-249, Feb. 1928.

caused solely be extrinsic pressure on a bronchus frequently shows fluctuating clinical findings. If sufficient outpouring of secretions and exudates fills the distal bronchioles and alveoli, the size of the involved pulmonary segment remains unchanged despite the presence of atelectasis and there results a "drowned" segment rather than a shrunken one.

A tuberculous process in hilar nodes may also lead to atelectasis by direct extension to an adjacent bronchus. The intensity of a tuberculous reaction is greater than that due to nongranulomatous pulmonary infections and there is frequent involvement of adjacent connective tissue, lung parenchyma, vessels, pleura, or bronchi. Penetrating caseation necrosis involving a bronchial wall leads to the development of concomitant endobronchial tuberculosis through the formation of fistulous tracts from the interior of the node. Masses of caseous or calcified debris may then be extruded into the bronchial lumen. Endobronchial ulcer formation or elevated granulomatous tissue may in themselves completely obstruct a bronchus or may sufficiently destroy the continuity of the ciliary and peristaltic motions of the bronchi so as to permit accumulation of secretions, bronchial obstruction, and atelectasis.

Brock *(24)* emphasized that a residual tight fibrous stricture frequently remains as a cause of bronchostenosis even after the active tuberculous involvement of a bronchus has apparently resolved. Once segmental atelectasis has occurred, future pathogenesis is determined by the duration of atelectasis and the presence or absence of infection. Usually the bronchi become filled with purulent exudate, with secondary destruction of the ciliated epithelium, submucosa, and, occasionally, of the entire bronchial wall. Focal pneumonitis may progress to widespread interstitial pulmonary fibrosis. Abscess formation and empyema are less common complications. Hennell *(25)* pointed out that the initiating tuberculous process in a bronchus may be completely submerged by the extensive secondary infection distal to the point of obstruction. Terminally there may remain an atelectatic, shrunken, bronchiectatic, and fibrotic pulmonary segment.

Fibrosis and bronchiectasis need not occur in every case, however, for it has been demonstrated that an atelectatic lobe may remain free of bronchiectasis for years if no infection occurs. All the clinical findings can then resolve completely if the bronchial obstruction is relieved *(5)*.

The special predilection of the right middle lobe to the development of atelectasis has been emphasized by recent writers *(13, 14, 24, 26-30)*.

(24) Brock, R. C.: Post-tuberculous bronchostenosis and bronchiectasis of middle lobe. Thorax 5: 5, 1950.

(25) Hennell, H.: Atelectasis as factor in evolution of chronic fibroid pulmonary tuberculosis. Am. Rev. Tuberc. 23: 461-475, May 1931.

(26) Bryant, J. R., and Harter, J. S.: Middle lobe disease. Dis. Chest 18: 250-253, Sept. 1950.

Brock (24, 27) explained this simply on the basis of the anatomy of the right hilar region. The right middle lobe bronchus is completely surrounded by lymph nodes which receive drainage from the middle and lower lobes of the right lung. In addition, the right middle lobe bronchus leaves the main stem bronchus at a very acute angle in which is consistently found a rather large lymph node. This exposes the right middle lobe bronchus to a greater risk of kinking or occlusion when compared to other major bronchi.

CLINICAL FEATURES

Chronic segmental atelectasis can occur at any age. The greatest number of reported cases have occurred in children. Sex distribution is of no apparent significance, although a recent series (30) of 8 female patients with an average age of over 60 years has been reported. In children, segmental atelectasis is frequently observed during the initial primary tuberculous infection. In adults, any acute bronchial or pulmonary infection can initiate this process, although the presence of previously diseased hilar lymph nodes makes the development of bronchial obstruction more likely. The exact cause in an individual patient may be difficult, if not impossible, to determine despite extensive diagnostic studies. Although fever, productive cough, chest pain, and abnormal physical findings are usually present at the onset of atelectasis, during the chronic phase of prolonged atelectasis the patient is rarely clinically ill. Recurrent pulmonary infection and a chronic cough with a variable degree of sputum production are common. Hemoptysis may occur. In short, a patient harboring an atelectatic segment frequently presents symptoms similar to those of minimal bronchiectasis.

Blood counts usually fall within normal limits after the atelectasis enters a chronic phase. Elevated sedimentation rates are present in most patients. The tuberculin skin test is almost always positive and careful study of the sputum and gastric washings with culture and guinea pig inoculation frequently gives conclusive proof of tuberculosis. The diagnosis of segmental atelectasis must be established by roentgenographic means, with special views and technics being employed as indicated. The exact site and cause of bronchial obstruction can often be visualized directly at bronchoscopy.

The duration of atelectasis is variable, lasting only a few weeks in some patients and remaining permanently in others. Kent (8) believes that if the atelectatic area re-expands within a period of 6 months the patient will usually escape bronchiectasis.

(27) Brock, R. C.: Anatomy of the Bronchial Tree. Oxford University Press, New York, N. Y., 1946. p. 52.

(28) Rubin, E. H., and Rubin, M.: Shrunken right middle lobe, with reference to so-called "middle lobe syndrome." Dis. Chest 18: 127-145, Aug. 1950.

(29) Zdansky, E.: Der Mittellappen als Punctum minoris resistentiae der Lunge. Wien. klin. Wchnschr. 58: 197-200, Apr. 19, 1946.

(30) Cohen, A. G.: Atelectasis of right middle lobe resulting from perforation of tuberculous lymph nodes into bronchi in adults. Ann. Int. Med. 35: 820. 1951.

ROENTGENOGRAPHIC FINDINGS

Although the diagnosis of atelectasis localized to individual pulmonary segments must be established roentgenographically, this is often difficult to accomplish because of several factors. The comparative roentgenographic density of normal and atelectatic pulmonary tissue is only slightly different; hence, an atelectatic area may be only faintly outlined and usually blends into normal tissue without definite demarcation. The cardinal signs of acute massive atelectasis (shift of the mediastinum, elevation of the diaphragm, narrowing of the rib spaces, and compensatory emphysema of the aerated lobes) are absent in segmental atelectasis. A collapsed segment or lobe shrinks medially toward the hilum and will not be seen in its usual location. Changes in contour or location of the interlobar septums, rearrangement of vascular shadows, and change in position of the hilum are important details to be noted in diagnosing lobar or segmental atelectasis.

A frontal view of the chest is often not striking and may appear almost normal. A lateral view is absolutely necessary for adequate study and visualization of the septal shadows. The importance of the lordotic view in a study of the middle lobe of the right lung and lingular segments of the upper lobe of the left lung is well documented. Additional special views may be indicated in some patients. Bronchograms may show failure of filling of the involved bronchial segment with compensatory displacement of surrounding bronchi, and occasionally bronchiectatic changes within the shrunken area may be demonstrated.

In a series of detailed papers, Robbins and Hale *(15-20)* have reviewed the diagnostic features of atelectasis in each of the various named bronchial segments.

BRONCHOSCOPIC FINDINGS

Bronchoscopy is of special importance in the study of chronic segmental atelectasis for in many patients the area of bronchial obstruction can be directly visualized. In addition, biopsy specimens and material for cellular study can be obtained which will aid in excluding the possibility of neoplasm. Bronchoscopic study of groups of children undergoing an initial tuberculous pulmonary infection reveals abnormal findings in a large percent *(4, 5, 9)*. These include (1) narrowing of the bronchial lumen due to extrinsic pressure presumably from an enlarged hilar lymph node, (2) ulcerated areas covered by granulation tissue, (3) endobronchial granulomatous masses, and (4) impending perforation of a bronchus by a tuberculous process in an adjacent node. In some patients, particles of caseous material can be removed from the bronchi.

Bronchoscopic findings in adults are essentially similar and include the occasional recovery of broncholiths or the direct visualization

of the granulomatous bed of a recently extruded broncholith (31). Cohen (30) regards the presence of anthracotic pigment in the vicinity of an old endobronchial scar as presumptive evidence of previous lymph node perforation. In patients in whom a frankly tuberculous lesion cannot be proved, narrowing of a bronchus with a slitlike orifice caused by mucosal edema and a nonulcerating inflammatory reaction are frequently found. Pus must sometimes be aspirated from a bronchus before such a condition can be visualized. Any of these bronchial lesions may terminate with a residual tight cicatricial stenosis. In some patients bronchoscopic study may fail to show the region of bronchial obstruction.

CASE REPORTS

Case 1. A 71-year-old retired mining engineer was hospitalized because of an arteriosclerotic aneurysm of the abdominal aorta. He had been previously studied in other hospitals because of atelectasis of the middle lobe of the right lung which remained roentgenographically unchanged over a period of several years. Bronchoscopy and repeated sputum cultures had produced only negative findings.

Physical examination revealed a senile, cachectic, critically ill man complaining of severe abdominal pain. A large pulsating mass was present in the lower abdomen; no abnormal findings were detected on examination of the chest. During the final hospitalization a localized infiltration developed in the left lower lung field along with a productive cough. Detailed additional study of the chest lesions was not possible because of the patient's critical condition. The abdominal aneurysm progressively increased in size with extensive terminal retroperitoneal hemorrhage from which the patient died.

Necropsy study showed complete atelectasis of the middle lobe of the right lung with extensive fibrosis and bronchiectasis. The bronchus was occluded by a group of enlarged firm encircling lymph nodes which yielded tubercle bacilli on culture and which showed fibrocaseous changes with surrounding areas of caseation necrosis on microscopic study. A small area of tuberculous caseation necrosis was also found in the lower lobe of the left lung.

Comment. Atelectasis in this patient was due to bronchial compression by enlarged tuberculous hilar nodes. The atelectasis was of undetermined duration and caused only mild symptoms and no disability during the period of observation. Although the tuberculous process in the hilar nodes was apparently quiescent, the patient's terminal debilitated condition caused it to become activated with tuberculous spread to an area in the left lung.

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(31) Fox, J. R., and Clerf, L. H.: Broncholithiasis; report of 10 cases. Ann. Int. Med. 23: 955-959, Dec. 1945.

Case 2. A 25-year-old housewife was originally hospitalized because of fever, cough, and sore throat. A roentgenogram of the chest led to an initial diagnosis of pneumonia and a course of aureomycin produced complete symptomatic relief within 3 days. The roentgenographic appearance of the lesion failed to change, however, and after additional views had been obtained, a diagnosis of atelectasis of the superior segment of the lower lobe of the right lung and calcification of the right hilar nodes was established (fig. 1). Bronchography revealed failure of filling of the involved segmental bronchus and bronchoscopy showed stenosis of the corresponding bronchus due to mucosal edema and inflammation. The patient was then transferred to this hospital where admission physical findings were normal throughout. The atelectatic segment remained unchanged roentgenographically on serial study. A first strength PPD tuberculin test was positive. The sedimentation rate was elevated to 32. Tubercle bacilli were eventually isolated from the sputum on both culture and guinea pig inoculation.

Case 3. A 22-year-old man was hospitalized because of an acute febrile illness with pain in the lower left portion of the chest and a nonproductive cough. Physical examination at that time revealed pharyngeal inflammation, diminished left thoracic expansion, decreased breath sounds, dullness to percussion, and inspiratory rales over the left lower posterior portion of the thorax. A roentgenographic diagnosis of calcification of hilar nodes and atelectasis of the anterior medial basal segment of the lower lobe of the left lung was made. Although penicillin and aureomycin administration resulted in rapid abatement of the acute symptoms, the cough became productive and the atelectatic area remained unchanged. A first strength PPD tuberculin test was positive. Sedimentation rates were elevated, averaging 35. Bronchoscopic examination was entirely normal. Gastric washings and sputum studies were eventually positive for tubercle bacilli on both culture and guinea pig inoculation. During the period of observation serial roentgenograms revealed only minimal lessening in the degree of segmental atelectasis.

Case 4. A 55-year-old woman was hospitalized because of low-grade fever, general malaise, and a feeling of tightness in the right side of the thorax, all of which had developed insidiously 2 months prior to admission. Her husband had been previously hospitalized for a total of 4 years because of active pulmonary tuberculosis. Routine roentgenograms of the patient's chest during that time had always been interpreted as negative, although a large group of densely calcified right hilar nodes were present. A roentgenogram of the chest on admission revealed an area of segmental atelectasis involving the apical and anterior segments of the upper lobe of the right lung. The sedimentation rate was persistently elevated, averaging 45, and the second strength PPD tuberculin test was positive. The right upper lobe bronchus showed mucosal edema and purulent secretions on

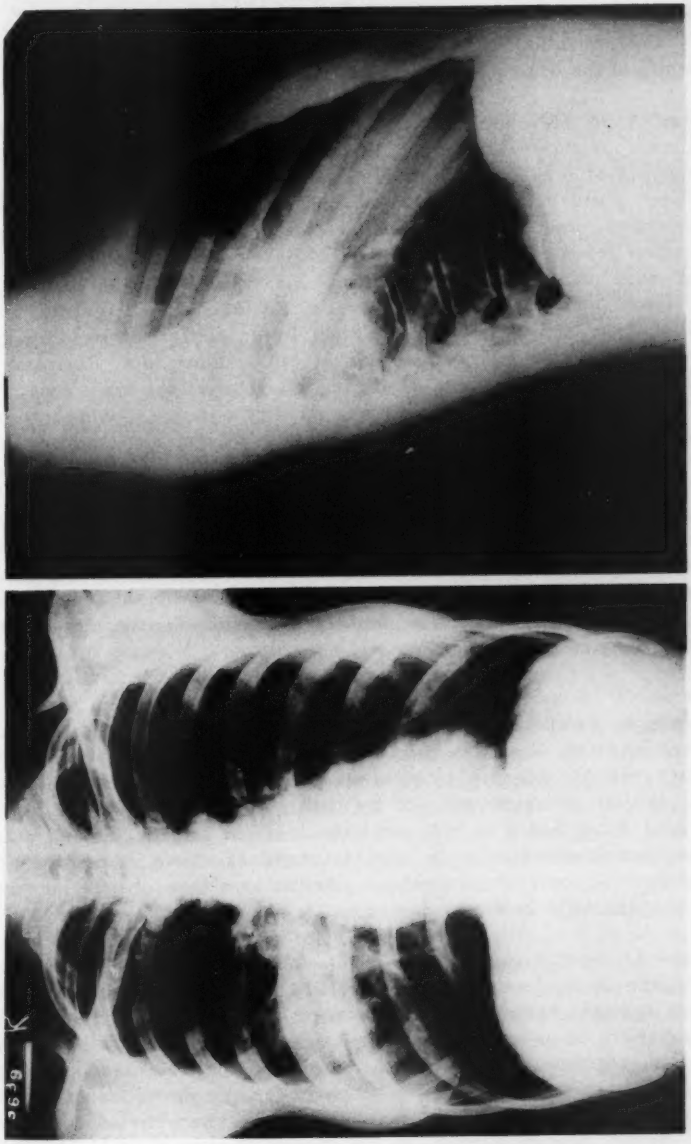

Figure 1 (case 2). Posteroanterior and lateral roentgenograms of the chest showing atelectasis of the apical segment of the lower lobe of the right lung.

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bronchoscopic examination. Gastric washings were positive for tubercle bacilli on both culture and guinea pig inoculation. During 2 months' hospitalization the patient was afebrile and became essentially asymptomatic. Serial roentgenograms of the chest showed slight lessening of the degree of atelectasis.

Case 5. A 56-year-old housewife was hospitalized because of a persistent productive cough of 18 months' duration and intermittent hemoptysis of 6 months' duration. Several brief courses of penicillin had been given by local doctors on the basis of a working diagnosis of recurrent bronchitis or bronchiectasis. Physical examination on admission revealed a slight lag of the right posterior portion of the chest with diminished breath sounds and scattered rales over the right lower posterior and lateral portion. Roentgenograms revealed an area of increased density in the right lower portion of the thorax, initially thought to represent interlobar effusion but later interpreted as indicative of atelectasis of the middle lobe of the right lung. A first strength PPD tuberculin test was strongly positive. Sedimentation rates were persistently elevated, averaging 38. On bronchoscopy, a friable granulating mass was seen at the bifurcation of the right middle and lower lobe bronchi. Biopsy from this mass was microscopically typical of tuberculous granulation tissue. This was confirmed by the isolation of tubercle bacilli from the sputum on culture. During a period of observation the atelectatic area remained roentgenographically unchanged. Follow-up reports after prolonged care in a sanatorium indicated that the tuberculous process was disseminated throughout the right lung.

Comment. A tuberculous cause was definitely proved in these 4 patients by culture. A different pulmonary segment was atelectatic in each instance. Case 4 is of interest in that the patient had prolonged contact with an actively tuberculous patient and had been followed with periodic roentgenograms of the chest, all of which showed large calcified hilar nodes in the strategic location for the development of segmental atelectasis. In case 5, atelectasis was caused by an endobronchial mass of tuberculous granulation tissue obstructing the orifice of the right middle lobe.

Case 6. Several months prior to his visiting the outpatient clinic a 56-year-old man, who had been retired from the Army because of severe cervical osteoarthritis, developed a persistent postnasal drip accompanied by a productive cough. Roentgenograms of the chest revealed complete atelectasis of the middle lobe of the right lung and calcification of right hilar lymph nodes. On hospitalization for diagnostic study, occasional inspiratory rales were heard over the right lower anterior portion of the thorax. Sputum examination was persistently negative for tubercle bacilli or atypical cells. A first

strength PPD tuberculin test was positive. Bronchoscopy showed no abnormalities. The patient continued to produce 30 cc. of sputum daily for several weeks. Serial roentgenograms revealed gradual clearing of the atelectasis with complete re-expansion of the lung occurring over an 8-week period. Follow-up studies have shown no recurrence of atelectasis.

Case 7. A 39-year-old sergeant was hospitalized because of the sudden onset of sharp pain in the anterior portion of the chest on the right side, productive cough, and low-grade fever, all of 1 week's duration. Physical examination revealed occasional fine rales over the lower half of the thorax anteriorly and posteriorly on the right side. An initial leukocyte count was 20,600 with 78 percent neutrophils and the sedimentation rate was 38. Roentgenograms were initially interpreted as showing an inflammatory reaction in the medial basal segment of the lower lobe of the right lung and a calcified node in the hilum on the right side. Serial films showed the development of marked atelectasis of the involved segment. The initial low-grade fever and purulent, occasionally blood-streaked sputum promptly disappeared following intramuscular injections of penicillin, postural drainage, and liquefying expectorants.

Sputum specimens were negative for tubercle bacilli or atypical cells. Bronchoscopy revealed no abnormality. A PPD tuberculin test was positive in the first strength. Bronchograms failed to fill the atelectatic segment and did not demonstrate bronchiectasis. The atelectatic segment showed gradual, although still incomplete, re-expansion during a 3-month period of observation and the patient was eventually permitted to return to military duty.

Case 8. A 46-year-old officer was hospitalized because of fever, chest pain, productive cough, and roentgenographic evidence of atelectasis of the middle lobe of the right lung with enlarged calcified hilar lymph nodes on the right side (fig. 2). Three years previously the patient had had a right-sided pneumococcic pneumonia with hemoptysis and residual chest pain. On entry occasional respiratory wheezes were heard over the thorax anterolaterally on the right side. Bronchoscopic findings on 3 occasions revealed narrowing of the orifice of the right middle lobe bronchus with diffuse mucosal edema and a profuse white exudate. Papanicolaou smears of the sputum were negative. Repeated studies of the sputum and gastric washings failed to reveal the presence of tubercle bacilli. The sedimentation rate on admission was 40. Several courses of penicillin and aureomycin were administered along with the use of postural drainage and liquefying expectorants. Over a period of 3½ months there was a gradual disappearance of symptoms along with clearing of the atelectasis on serial roentgenograms. The patient was eventually returned to full military duty. Follow-up examination after 14 months showed no recurrence of atelectasis.

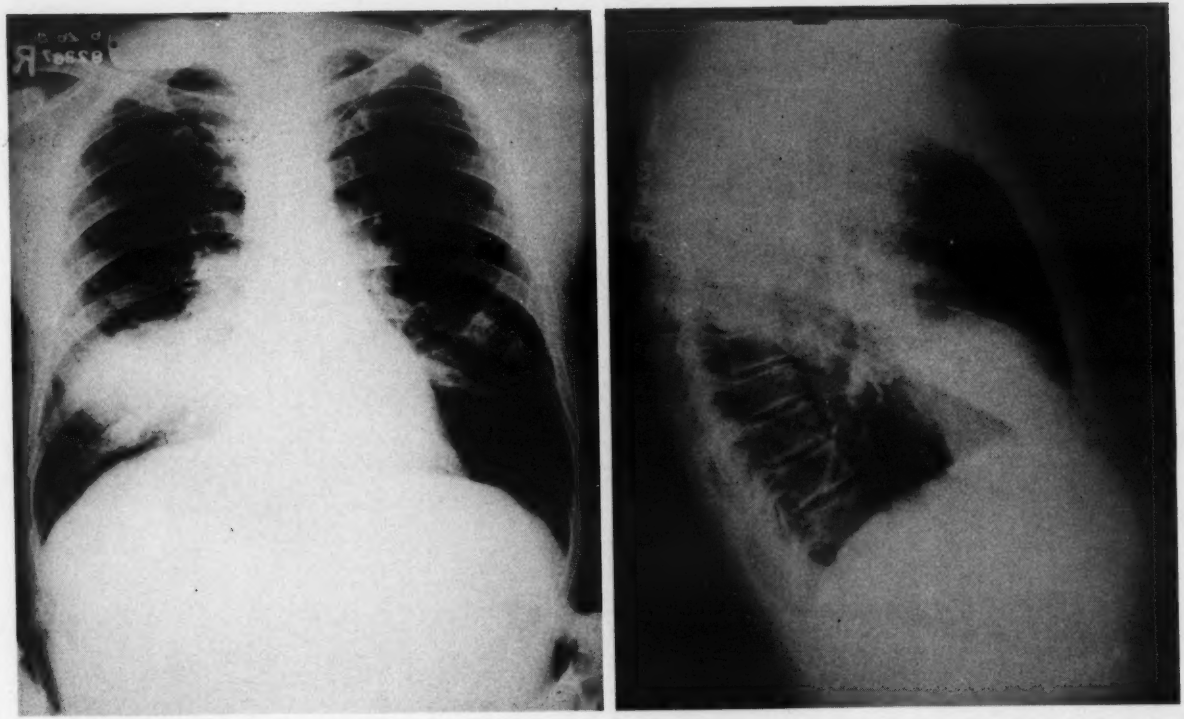

Figure 2 (case 8). Postero anterior and lateral roentgenograms of the chest showing atelectasis of the middle lobe of the right lung.

Case 9. A 47-year-old housewife was hospitalized because of fever, cough, dyspnea, and general malaise, which had developed over a period of several weeks. Occasional rales and increased vocal fremitus over the thorax anteriorly on the right side were present on admission, along with a diastolic apical heart murmur typical of mitral stenosis. On radiographic study the middle lobe of the right lung was seen to be atelectatic. The cardiac contour was suggestive of early mitral valvular heart disease and calcified hilar nodes were present. The sedimentation rate was elevated to 28. Bronchoscopy revealed edema and inflammation of the entire tracheobronchial tree, most marked in the region of the right middle lobe bronchus which was almost completely occluded. Papanicolaou smears from the bronchi were negative. Repeated sputum studies and gastric washings were negative for tubercle bacilli. Symptoms disappeared without specific therapy and serial roentgenograms showed slow, progressive resolution of the atelectatic area with complete clearance in 3 months. A second bronchoscopy performed 6 weeks after the first was entirely normal.

Comment. Despite careful diagnostic study of these 4 patients a cause could not be conclusively established, but in each instance there was roentgenographic demonstration of a calcified hilar node in the strategic location for compression of the involved segmental or lobar bronchus. Re-expansion of the atelectatic segments occurred after a period of from 6 weeks to 3½ months.

Case 10. A 17-year-old soldier was transferred to this hospital because of residual segmental atelectasis in the lower lobe of the right lung following an acute pneumonitis which had been treated with penicillin. On admission he was asymptomatic and the physical findings were normal throughout. Bronchoscopy was normal. Bronchography demonstrated failure of filling of the anterior basal segment of the lower lobe of the right lung. A PPD tuberculin test was positive in the first strength. There was no sputum production and gastric washings were negative for tubercle bacilli. Despite prolonged observation, serial roentgenograms of the chest remained unchanged. It was believed that surgical exploration was not warranted and the patient was returned to full military duty. Shortly thereafter he reported to another Army hospital, complaining of vague pain in the right side of his chest and a nonproductive cough. Bronchoscopy then revealed some hyperemia of the right lower lobe bronchi. Gastric washings were again negative for tubercle bacilli and the patient was eventually returned to full military duty with no cause for the chronic atelectasis having been demonstrated. Roentgenograms taken 18 months after the onset of the atelectasis continued to show a minimal radiographic density in the anterolateral basal segment of the lower lobe of the right lung.

Case 11. A 32-year-old sergeant was initially hospitalized because of nervousness, productive cough, and the loss of 40 pounds. He had

2 previous episodes of pneumonia and had noted occasional streaks of blood in his sputum. Roentgenograms revealed atelectasis of the middle lobe of the right lung which remained unchanged on serial study (fig. 3). Bronchoscopy revealed a friable, bleeding mass at the orifice of the right middle lobe bronchus and the patient was transferred to this hospital with diagnosis of probable bronchiogenic carcinoma.

Physical examination revealed dullness and decreased tactile fremitus in the right axilla. A first strength PPD tuberculin test was positive. Repeated sputum studies and gastric washings failed to demonstrate tubercle bacilli or malignant cells. A specimen from the previously described intrabronchial mass was examined microscopically on 3 occasions with an interpretation of chronic inflammation on 2 occasions and of granulomatous reaction consistent with tuberculosis on the third. The patient was then transferred to Fitzsimons Army Hospital. A bronchogram failed to fill the right middle lobe bronchus but was otherwise negative. An exploratory thoracotomy was performed and the middle lobe of the right lung was found to be atelectatic and firm. Enlarged hilar lymph nodes were found compressing the right middle lobe bronchus. As the middle lobe was being resected, thick white pus escaped from the bronchus. A tissue diagnosis of atelectasis of the middle lobe of the right lung with secondary suppurative, ulcerative bronchitis was made. Changes in the lymph nodes were nonspecific microscopically and a tuberculous cause could not be established by culture. Following operation the patient made an uneventful recovery and was returned to full military duty.

Case 12. A 28-year-old housewife was hospitalized because of persistent atelectasis of the middle lobe of the right lung which had been present roentgenographically for about 11 months. A calcified primary complex with enlarged calcified right hilar nodes was also present. This atelectasis had originally developed during a brief episode of pain in the right anterior portion of the chest, fever, and cough. The cough remained constant and productive, accompanied occasionally by raising bright red blood in amounts up to 200 cc. Despite this atelectasis, the patient had gone through an uneventful and successful full-term pregnancy.

No abnormal physical findings were detected on hospitalization. Repeated studies of the sputum failed to demonstrate tubercle bacilli and Papanicolaou smears of the sputum were negative. The sedimentation rate was 15. Bronchoscopy was negative on 2 occasions. Initial bronchograms revealed complete collapse of the middle lobe of the right lung with failure of filling of any of its bronchi. A second bronchogram performed 1 year after the first revealed that some lipiodol had entered the bronchus to the lateral segment and had proved it to be bronchiectatic. The patient refused an exploratory thoracotomy. Productive cough, occasional hemoptysis, and right middle lobe atelectasis have persisted for a period of 2½ years of observation despite medical management.

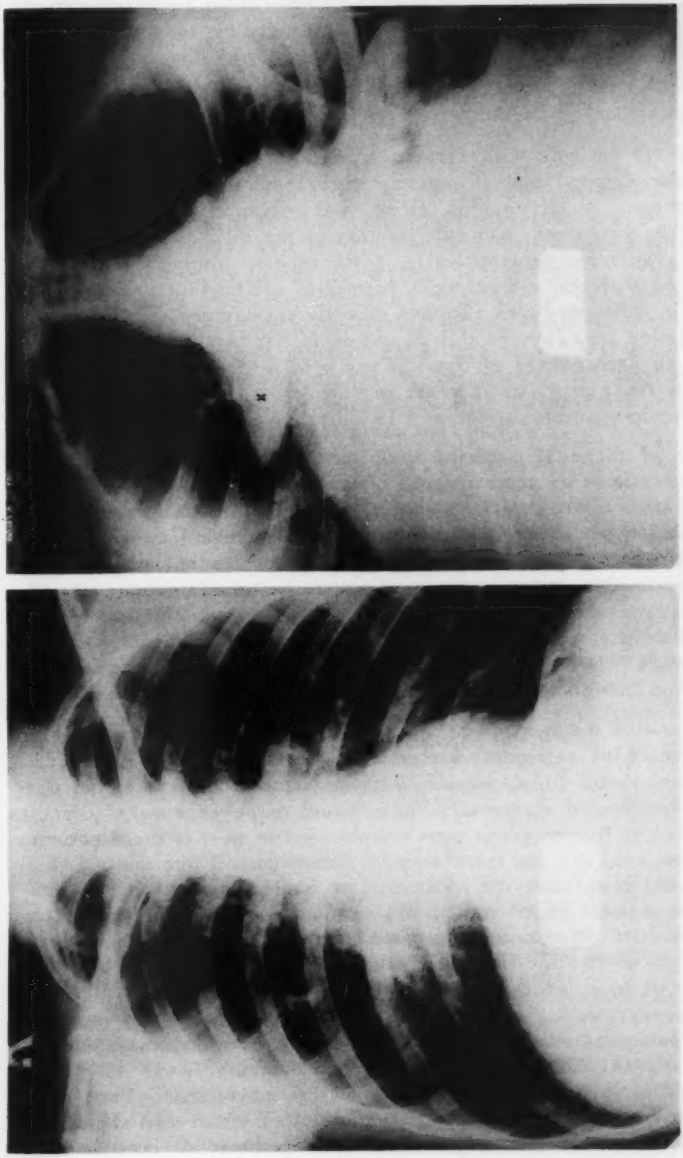

Figure 3 (case 11). Posteroanterior and lordotic roentgenograms of the chest showing the value of the lordotic view in demonstrating atelectasis of the middle lobe of the right lung.

Comment. Segmental atelectasis in these 3 patients was unchanged on prolonged observation. No specific cause could be demonstrated. In cases 10 and 11 an operation was recommended because of persistent symptoms. This was carried out in case 10 with the finding of an atelectatic fibrotic lobe caused by pressure from enlarged hilar nodes.

DISCUSSION

These 12 patients with chronic segmental atelectasis demonstrate the major clinical and roentgenographic features noted in previous publications. A tuberculous cause was definitely established by culture in 5 patients, and calcification of the hilar lymph nodes has been shown roentgenographically in the others. Although the largest series of patients with chronic segmental atelectasis has been reported in children, Brock *(24)* has recently emphasized the importance and relative frequency of this condition in adults and Cohen *(30)* reported an average age of over 60 years in his series of 8 patients. The age range of 17 to 71 years in this series bears out the opinion that chronic segmental atelectasis is a disease of adults.

Eight of these 12 patients were able to date the apparent onset of atelectasis to an acute episode of bronchial or pulmonary infection. The duration of atelectasis has been prolonged in each instance, with re-expansion occurring in no shorter than 6 weeks in any of these patients. Four additional patients have been studied who are not included in this series because of a duration of atelectasis of only about 3 weeks. After an area of segmental atelectasis had become well established the predominant symptom was chronic, usually productive cough. These patients usually appeared to be in good general health and showed no abnormal physical findings.

The routine roentgenograms were usually inadequate in establishing a diagnosis of segmental atelectasis. Detailed study with special views and serial films, together with constant awareness of the possible presence of atelectasis, were found to be necessary adjuncts to diagnosis. Bronchograms were not obtained in most of these patients as we believed that the instillation of radiopaque oil into the bronchial tree would complicate the evaluation of future serial films. The atelectatic segment failed to fill in 4 patients on whom bronchography was performed. A second bronchogram obtained in case 11 (1 year after the first) showed some filling of the involved bronchus which was then seen to be bronchiectatic. Bronchograms were not obtained following spontaneous clearing of the atelectasis in any patient. Bronchoscopy was of paramount importance, especially in ruling out malignant lesions. Two patients showed a granulating endobronchial mass which, on microscopic examination, appeared to be tuberculous. Five other patients had narrowing of the bronchial lumen which was thought to be due to mucosal edema. In 5 patients the bronchoscopic findings were normal throughout. A diagnosis of bronchial compression due to extrinsic pressure could not be established bronchoscopically in any of these patients.

During the period of prolonged observation and study of these patients, therapy was essentially conservative, consisting largely of symptomatic measures, occasional courses of penicillin or aureomycin. In only 2 patients was operation advised. This was refused by 1 patient and resulted in complete cure in the other. A conservative approach to operation in these patients was advised by Brock *(24)* and Cohen *(30)*. Rubin and Rubin *(28)* also believe that operation is indicated only in patients having frequent pneumonia, recurrent hemoptysis, or superimposed suppuration. Brock warned of possible postoperative complications from tuberculous hilar lymphadenopathy activated by operation, and Rubin and Rubin suggested the preparation of every patient with segmental atelectasis with a course of streptomycin before attempting excision. Streptomycin therapy is condemned, however, in patients in whom a definite tuberculous cause has not been established by culture and in whom no operation is contemplated. Antibiotics which have no effect on tubercle bacilli are of value in reducing or eliminating an infectious process in the bronchi and may lead to reduction of mucosal edema, elimination of purulent bronchial secretions, and to re-expansion of the atelectatic area.

SUMMARY

Chronic segmental atelectasis is relatively common. The probable tuberculous cause of chronic segmental atelectasis is stressed with the finding of calcified hilar lymph nodes in all and the isolation of tubercle bacilli in 5 of a series of 12 patients. Special roentgenographic views, serial films, and bronchoscopy are of value in the diagnosis and study of chronic segmental atelectasis. In the medical management of these patients (when tuberculous cause is unproved) the probability of an underlying tuberculous process must be kept in mind. Surgical therapy should be reserved for those patients showing frequent pneumonia, recurrent hemoptysis, or superimposed infection.

BOOK REVIEWS

Rehabilitation Nursing, by *Alice B. Morrissey,* B. S., R. N., Instructor in Rehabilitation Nursing, New York University-Bellevue Medical Center; Supervisor of Nursing Service, Department of Physical Medicine and Rehabilitation, Bellevue Hospital. Foreword by *Howard A. Rusk,* M. D., Professor and Chairman of the Department of Physical Medicine and Rehabilitation, New York University-Bellevue Medical Center; Associate Editor, The New York Times. 299 pages; illustrated. G. P. Putnam's Sons, New York, N. Y., publisher, 1951. Price $5.

The author here describes the meaning of rehabilitation and the important role the rehabilitation team plays in modern medicine. The tendency to treat the disease and not the patient as a whole has been the trend of the past and this book brings to light the fact that the

modern nurse should have training and experience in rehabilitation. The chapter dealing with the care of the paraplegic and quadriplegic is informative and should be beneficial to those working with such patients. This book will aid the bedside nurse in realizing her many opportunities to help the patient before he is ready for medical referral to a special department for treatment.—*Lt. Comdr. A. Davidson, NC, U. S. N.*

Approved Laboratory Technic, by *John A. Kolmer,* M. D., D. P. H., Sc. D., F. A. C. P., Professor of Medicine and Director of the Institute of Public Health and Preventive Medicine, Temple University; Formerly Professor of Pathology and Bacteriology, Graduate School of Medicine, University; of Pennsylvania; and *Earle H. Spaulding,* Ph. D., Professor of Physiological Chemistry; Temple University School of Medicine. 5th edition. 1,180 pages; 403 illustrations; 28 color plates. Appleton-Century-Crofts, Inc., New York, N. Y., publishers, 1951. Price $12.

To have reached the fifth edition indicates the esteem in which this book is held. The methods described cover the whole range of procedures that the clinical laboratory might be called on to do. The technics are presented from the standpoint of the large, busy laboratory. The general pattern of presenting these technics is to describe the principle, followed by details of the procedure, and calculations where indicated. The inclusion of "sources of error" is a valuable feature. Normal values are given, but there is seldom any comment on clinical application. The chapters "Diseases of Animals and Methods for the Care, Inoculation, and Bleeding of Animals" and "Methods for the Prevention and Treatment of Laboratory Accidents" are especially interesting and the section on serologic methods is one of the outstanding sections of the book. Here, as elsewhere, it would be helpful to have some statement of choice among several procedures, or indication of situations in which one may be better than another.

There is remarkably little duplication, indicating careful editing. The periodic acid-Schiff technic for demonstrating fungi was included in the section on histologic technic, but was not mentioned in preparation of skin scrapings, et cetera under "Diagnostic Mycologic Methods."

Insufficient background material is presented to make this an especially useful text for students beginning the study of laboratory methods, but it is an invaluable aid to the trained laboratory worker.

—*Lt. Col. J. P. Tollman, U. S. A. F. (MC)*

Sterilization of Footwear[1]

John D. Fulton, *Major, U. S. A. F. (MSC)*

Roland B. Mitchell, *Ph. D.*

DERMATOPHYTOSIS (athlete's foot) is a major problem in both civilian and military medicine. Infections of this type can become severe enough to cause the loss of many man-hours. The number of patients with dermatophytosis is much higher than commonly realized. Fox and Shields (2) have shown that more than 50 percent of all men in the United States at one time or another are infected by this and related fungi.

In some patients modern medication and methods of therapy for these infections are highly successful, but complete cure usually requires prolonged treatment even under ideal conditions. In many patients, treatment is further complicated by constant reinfection due to the continued wearing of contaminated shoes and stockings. Berberian (3) showed that fungi remain viable in stockings after simple laundering. In a study of 100 patients with mild, recurring dermatitis, Jamieson and McCrea (4) recovered pathogenic fungi from 40 percent of the shoes and organisms of doubtful pathogenesis from an additional 34 percent. They believed that footwear may harbor pathogenic fungi and may be a source of reinfection as well as of primary infection. They advised the sterilization of footwear at intervals during the course of treatment.

The most common methods in use at present for sterilizing footwear consist of either immersion of the shoes in weak solutions of formaldehyde or pouring formaldehyde solution into the shoe and storing the shoe in a tight box (5). These methods, while comparatively simple, leave much to be desired as the shoes must be thoroughly cleaned and treated with leather preservatives to protect their wearing qualities after sterilization.

(1) U. S. Air Force School of Aviation Medicine, Randolph Field, Tex.

(2) Fox, E. C., and Shields, T. L.: Résumé of skin diseases most commonly seen in general practice. J. A. M. A. 140: 763-768, July 2, 1949.

(3) Berberian, D. A.: Dermatophytosis of feet; sources and methods of prevention of reinfection. Arch. Dermat. & Syph. 38: 367-372, Sept. 1938.

(4) Jamieson, R. C., and McCrea, A.: Ringworm of feet; shoes and slippers as source of reinfection; final report. Arch. Dermat. & Syph. 44: 837-844, Nov. 1941.

(5) Weidman, F. D.; Emmons, C. W.; Hopkins, J. G.; and Lewis, G. M.: War and dermatophytosis, with special reference to treatment and fungicide testing. J. A. M. A. 128: 805-811, July 14, 1945.

A gas that could penetrate all parts of the snoe, be harmless to the leather, and require no poststerilization treatment would be ideal. Greene (6) used formaldehyde vapor but found that it did not satisfactorily penetrate the cracks of the shoes. Traub and Schultheis (7) found that chlorine gas killed mycelia and spores in vitro. Hydrogen cyanide gas is an excellent fungicide but is toxic and dangerous to use without rigid safety precautions.

An ideal gas for sterilizing shoes would be highly fungicidal, easily removed by aeration, highly diffusible, and would act rapidly. In addition, it should be noninflammable, nonexplosive, and easy to obtain and store. Ethylene oxide gas seems to meet most of the above requirements (8). It has been used as a fungicide and an insecticide in the food and storage industries for several years. It is inflammable and the vapor forms explosive mixtures with air in all proportions from 3 to 80 percent. The explosive potentials are eliminated when the gas is mixed with about 8 times its volume of carbon dioxide (9). A commercial mixture of 10 percent ethylene oxide and 90 percent carbon dioxide (carboxide) is available and was used in our investigation.

The initial work was accomplished using a complicated exposure chamber. Although satisfactory for the preliminary studies, this chamber would be unsatisfactory for use under field conditions where suitable materials and proper tools for its construction might be difficult to obtain. A satisfactory chamber should be simple to construct or to adapt from scrap material without requiring complicated tools. After considering numerous ideas, materials, and methods for the construction of such exposure chambers it was decided that the most satisfactory and simple types would be those that could be constructed from available drums, cans, airtight bags, or sacks. With this in mind, tests were made on chambers constructed from a 25-gallon galvanized iron (G. I.) can, a 5-gallon hypochlorite bleach can, a standard quartermaster methyl bromide delousing bag, and a rubber inner tube.

EXPOSURE CHAMBERS

Wooden box. This exposure chamber measuring 10 by 10 by 20 inches (fig. 1) was constructed from 3/4-inch plywood. The gas inlet was located in the center of the left side, 1 inch from the bottom and was equipped with a valve to control the flow of gas. The gas and air outlet valve was located in the center of the right side, 1 inch from the top. The door of the chamber was hinged on one side and equipped with 3 spring catches on the opposite side. These catches were designed as

(6) Greene, H. S.: Report on microbiological flora and sterilization of used shoes. J. Am. Leather Chem. 40: 96. Mar. 1945.

(7) Traub, E. F., and Schultheis, D., Jr.: Gassing of fungi; preliminary report. Arch. Dermat. & Syph. 60: 272-276, Aug. 1949.

(8) Phillips C. R., and Kaye, S.: Sterilizing action of gaseous ethylene oxide; review. Am. J. Hyg. 50: 270-279, Nov. 1949.

(9) Jones, G. W., and Kennedy, R. E.: Extinction of ethylene oxide fumes with carbon dioxide. Indust. & Engin. Chem. 22: 146-147, 1930.

a safety mechanism to prevent possible damage to the chamber in case of sudden pressure increase and yet to hold the door sufficiently tight to minimize escape of the gas. A thin felt gasket was glued to the door to insure a tight fit. A manometer was placed on the inlet side of the chamber to serve as an aid in observing and calculating the gas flow.

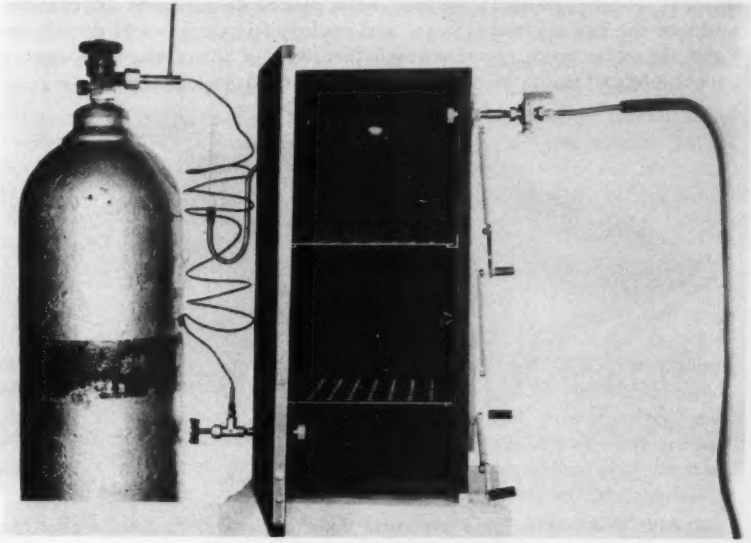

Figure 1. Wooden box exposure chamber.

Twenty-five gallon G. I. can. A felt gasket ½-inch thick and 2-inches wide, was glued to the inside of the can lid to form a seal between the lid and the rim of the can. A Schrader valve was soldered into the side of the can about 2 inches from the bottom. A 1/4-inch brass cock valve was soldered into the lid of the can as an outlet port. As the gas entered the can from the bottom inlet valve it displaced the air upward, forcing it through the outlet valve in the lid of the can, thereby filling the can with gas.

Hypochlorite bleach drum. This drum, containing a hypochlorite bleach for decontaminating, was originally issued by the Chemical Corps. It had a volume of 2.2 cu. ft. and a diameter of about 14 inches, and a fully removable gasketed head with a draw-type head-retaining clamp. It was originally designed as an airtight container for hypochlorite slurry. A piece of gum rubber tubing with an outer diameter of 5/16 inch was adapted as a gasket to insure an airtight seal. A Schrader valve was soldered into the side of the drum about 2 inches from the top for an inlet valve. A 1/4-inch brass cock valve was soldered into the drum head as an outlet valve. Tests made on this drum indicated that it was airtight and would withstand low pressures.

Delousing bag. A standard quartermaster methyl bromide delousing bag made of rubberized fabric was fitted with a Schrader valve as a gas inlet port. A clamp, consisting of 2 pieces of ½-by 4-inch plywood sufficiently long slightly to overlap the bag, was devised. One-half inch holes were drilled every 5 inches through the center of both strips. Machine bolts, 3/8 by 3 inches, were placed through the holes. The mouth of the bag was folded over and enclosed in the jaws of the clamp. When the bolts were tightened and the jaws of the clamp compressed over the folded mouth of the bag an airtight seal was formed.

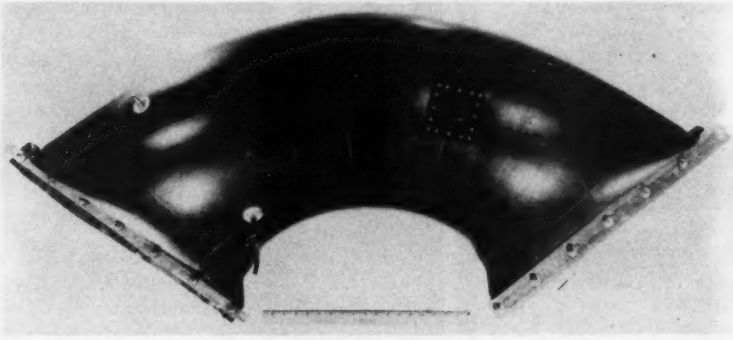

Figure 2. B-29 inner tube chamber.

Rubber inner tube. This exposure chamber was made from a one-third segment of a used B-29 aircraft inner tube. A 4-inch strip from one end of the tube was sanded and sealed together with rubber cement. A clamp made from 2 metal bars (1 by 3/4 inch), sufficiently long to overlap the sides of the tube was applied to the sealed end. Seven holes, ½ inch in diameter, were drilled in the bars and through the sealed portion of the tube. Machine bolts, 3/8 inch in diameter, were placed through the holes and, when tightened, formed an airtight seal. The other end of the tube was fitted with a metal clamp similar in pattern with that used on the delousing bag. Two Schrader valves were fitted into the wall of the tube as inlet ports for the gas. A plexiglass inspection port was constructed in the wall of the tube. When the mouth of the bag was fastened with the clamp, an airtight chamber that could withstand slight pressures was obtained. The volume of this chamber when filled with air to a pressure of 20 cm. of water was 120 liters (fig. 2).

SAMPLING TECHNIC

Sampling technics were developed to determine the efficacy of the treatment. In the technic first used, a sterile ½-inch leather punch was placed in the chuck of an arbor press. By using the press, sufficient pressure was obtained to drive the punch completely through the toe of the shoe thereby punching out ½-inch diameter samples from all layers of the shoe. The various lavers of the shoe were then separated

aseptically and plated on Sabouraud's agar. The punch and the punching area were surrounded with a protective tent of sterile towels saturated with a 5-percent phenol solution as an additional protective measure.

While this sampling technic was used it was observed, in numerous cases, that the cork inner liner of the treated shoes showed growth, whereas the other layers of the shoe were apparently sterile. This could be due to the impermeability of this particular layer to the ethylene oxide. As sterilization of those areas of the shoe that come in direct contact with the foot was of primary interest, it was possible that complete sterilization of all layers of the shoe would not be necessary if micro-organisms located in the inner layers would not migrate to the layers that come in contact with the foot. If this were so, the punch sampling technic was a much too rigid a test. A technic which would show whether the layers that came in contact with the foot were sterile would suffice.

Based on this hypothesis, the following sampling method was developed: The toes of the shoes were cut from the rest of the shoe with a band saw and both portions of the shoe were placed in the exposure chamber. Only the toe of the shoe was used for sterility determinations, but both portions of the shoe were exposed to the ethylene oxide gas. This was done to insure that the quantity of gas absorbed during treatment would be equivalent to that absorbed by a whole shoe. After treatment with the gas the shoe toes were removed aseptically from the container and placed in sterile cellophane bags for aeration. On completion of the proper periods of aeration the mouth of the cellophane bag was opened and 20 cc. of melted sterile Sabouraud's agar (51° C.) were poured into the inside of the shoe toe. The shoe toe was then rotated as the agar solidified, thereby coating the entire inner surface of the shoe toe with a layer of agar about 1/8-inch thick. To prevent drying during incubation, the shoe toes were placed on moist pads of sterile filter paper in large culture dishes. Incubation of these plates was accomplished in a humidified incubator at 37° C.

EXPERIMENTAL METHODS AND RESULTS

Strips of leather about 1/4 by 1/4 by 1/2 inch were cut from a nonsterilized shoe sole. To test for the presence of viable organisms some of the strips, selected at random, were planted on Sabouraud's agar in Petri plates. The plates were incubated for 10 days at room temperature. Micro-organisms were recovered from all of the nonsterilized leather samples. Organisms recovered by this procedure were identified as various species of the *Aspergillus, Penicillium, Mucor, Rhizopus, yeast, Bacillus subtilis,* and others not so readily identified. Other leather samples cut from the same shoe were placed in a 250 ml. cotton-plugged Erlenmeyer flask and placed in the box chamber. The inlet and outlet valves on the chamber were opened and the carboxide gas was allowed to flow into the chamber. The flow of gas was sufficiently long to insure that all of the air in the chamber was replaced by the gas

(about 2 minutes). The cylinder valve was then closed and both of the chamber valves were closed to prevent escape of the gas. This procedure was carried out at 0800 and at 1700 for 2 days. Between gassings the door of the box remained closed. At the end of 48 hours the samples were removed aseptically and plated on Sabouraud's agar. The plates were then incubated for 10 days at room temperature. Microorganisms were not recovered from any of the sterilized samples.

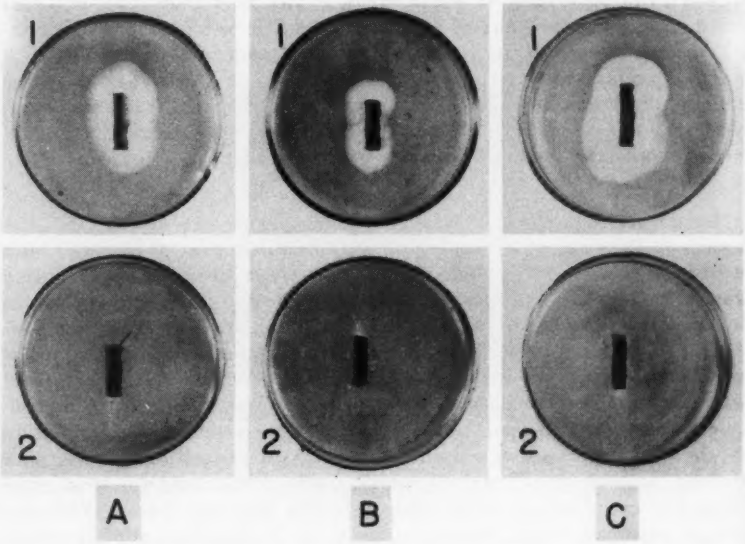

Figure 3. Sterilized leather samples contaminated with pathogenic fungi. (A) M. gypseum. (B) M. canis. (C) T. mentagrophytes. *Specimens numbered* 1 *were nonsterilized; those numbered* 2 *were sterilized.*

The next series of experiments was designed to determine the possibility of sterilizing leather samples contaminated with certain pathogenic fungi. For the initial tests *Microsporum canis, Microsporum gypseum,* and *Trichophyton mentagrophytes* were used as test organisms. These fungi were planted on Sabouraud's agar in Petri plates and incubated at 37° C. for 16 days. Leather samples previously sterilized with carboxide gas were then pressed into the growing fungus colony. The samples remained on the colony for 12 days at 37° C., at which time the leather samples were completely overgrown with the fungus. At the end of this period, some of the contaminated samples, selected at random, were planted on Sabouraud's agar plates and incubated at 37° C. for 10 days. The remaining contaminated samples were placed in dry, sterile Petri plates and exposed to carboxide gas for 48 hours. At the termination of exposure the samples were aerated, planted on Sabouraud's agar and incubated at 37° C. for 15 days.

No fungus growth was obtained from any of the treated samples contaminated with the test organisms but luxuriant growth was obtained from each of the nontreated contaminated samples (fig. 3).

To determine the possibility of sterilizing other pathogenic fungi by exposure to carboxide gas in the delousing bag chamber, cultures of *Monosporium apiospermum, Phialophora verrucosa, Epidermophyton floccosum, Sporotrichum schenckii, Trichophyton rubrum,* and *Trichophyton schoenleini* were plated on Sabouraud's agar in Petri plates. These cultures were incubated at 37° C. for 16 days. Leather samples cut from shoe soles and previously sterilized by exposure to carboxide gas were pressed into the growing fungus colonies and allowed to remain for 12 days at 37° C. At the end of this period the contaminated samples were removed and placed in dry sterile Petri plates. Some of the plates containing the contaminated samples were placed in the delousing bag chamber and the bag was filled to tautness with carboxide gas. Eight hours later the chamber was regassed to the original degree of tautness. Twenty-four hours after the original application of gas the plates containing the samples were removed and aerated for 6 hours. The samples were then planted on Sabouraud's agar and incubated for 15 days at 37° C. The remaining contaminated samples were plated directly, without further treatment, on Sabouraud's agar and incubated with the treated samples. At the termination of the incubation period all of the samples treated with carboxide gas showed no growth of fungi, and the controls all showed fungus growth.

STERILIZATION OF SHOES

G. I. can. Well-worn shoes, wrapped in paper to prevent recontamination during later handling, were placed in the can. The carboxide gas was turned into the can through the bottom inlet valve and allowed to flow for about 2 minutes. The chamber was regassed at 1, 3, 5, 6, 21, and 23 hours after the initial gassing. At the termination of the experiment (24 hours) the shoes were removed from the can, aerated in their paper wrappings for 3 hours, and sampled by the punch technic. The layers obtained were separated and plated on Sabouraud's agar and incubated for 7 days at 37° C. The mates to the treated shoes were sampled directly and plated. At the end of 7 days' incubation each of the control or untreated shoes showed fungus growth from all layers. Two of the treated shoes gave no growth from any of the layers, but the other treated shoes showed growth from all of the layers. This difference might be explained by the position of the shoes in the container. The shoes that were closer to the bottom of the container may have been exposed to higher concentrations of ethylene oxide; however, no records concerning this were available. As the results obtained with this container were not sufficiently good to justify further work, it was decided to test another of the exposure containers.

Hypochlorite bleach drum. Four well-worn shoes were wrapped in paper and placed in the container. The drumhead was tightened and

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the chamber was filled with gas. Carboxide gas was allowed to flow slowly into the container from the cylinder for about 5 minutes, in order to insure displacement of the air in the chamber. The outlet valve was closed at the end of this time and the container was brought to a pressure of 5 pounds per square inch with the gas. Twenty-four hours later, immediately prior to removal of the shoes, it was found that the pressure had dropped to 3.12 pounds per square inch. This loss of pressure could have been caused by slow diffusion of the gas through the rubber seal and diffusion of the gas into the shoes. The shoes were removed from the chamber and aerated in their paper wrappings for 6 hours. Samples were then punched from the toes of the shoes. The leather layers were separated and plated on Sabouraud's agar. The plates were incubated for 6 days at 37° C. At this time fungus growth was observed on all of the treated samples as well as the control samples. The experiment was repeated and identical results were obtained. It was not readily apparent why the shoes exposed to the gas in this container were not sterilized. Because of the poor results obtained no further tests were made using this container.

TABLE 1. *Results of exposure to carboxide gas for 24 hours in the delousing bag chamber*

Shoe number	Layer of shoe				
	Outer layer	Inner layer (top)	Inner sole	Cork inner liner	Outer sole
9	-	-	+	+	+
3	+	+	+	+	+
4	-	-	-	+	-
24	-	-	-	+	-
2	-	0	-	+	0
11	-	-	-	+	0

+ = growth
- = no growth
0 = not recorded

Methyl bromide delousing bag. Six unwrapped, well-worn shoes were placed in the bag, and the mouth of the bag was closed by applying the clamps. The bag was then filled to tautness with carboxide gas. Eight hours after the original application of the gas it was observed that the bag was partially deflated. This loss could have been caused by diffusion of the gas through the walls of the bag and by diffusion into the shoes. Additional gas returned it to its original degree of inflation. At the end of 24 hours the shoes were removed aseptically from the bag and placed in sterile paper sacks, aerated for 6 hours, and sampled by the punch technic. The samples obtained were plated on Sabouraud's agar. During previous experiments it had been observed

that certain layers of the shoe were more difficult to sterilize than others. The one layer that consistently showed fungus growth was the cork inner liner. To obtain more information relative to this a record was kept of the location of the various layers on the plate. The plates were incubated at 37° C. for 6 days. The mates to the treated shoes were sampled and plated as controls. As can be seen in table 1 certain layers showed no growth following treatment, but the cork inner liner gave growth in all samples. Fungus growth was obtained from *all* layers in each of the untreated control shoes.

TABLE 2. *Results of exposure to carboxide gas for 48 hours in the delousing bag chamber*

Shoe number	Layer of shoe				
	Outer layer	Inner layer	Inner sole	Cork inner liner	Outer sole
16	-	-	-	+	-
7	-	-	-	+	-
18	-	-	-	+	-
15	-	-	-	-	-
22	-	-	-	-	-
17	-	-	-	-	-

+ = growth
- = no growth

In the light of these results it was deemed advisable to repeat the experiment exposing the shoes to the gas for 48 hours instead of the 24 hours. Six shoes were placed in the bag and the bag was filled with carboxide to tautness at 0, 7, 22, 31, and 46 hours. After 48 hours the shoes were removed and aerated for 6 hours in sterile paper bags. The shoes were sampled and plated on Sabouraud's agar. The mates to the exposed shoes were sampled and plated as controls. After an incubation period of 7 days at 37° C. *all* of the layers from each control shoe showed fungus growth. Three of the treated shoes were completely sterile and fungus growth was obtained from the cork inner liner only of the remaining 3 shoes (table 2). The results of this experiment were quite satisfactory, but 48 hours' exposure time is much too long for a satisfactory rapid method of sterilization.

Throughout the course of the tests using the delousing bag as an exposure chamber it was observed that a great deal of gas was lost by diffusion through the walls of the bag. This gradual loss would tend to reduce the concentration of ethylene oxide below that level required for sterilization. To reduce this loss, thereby maintaining a higher concentration of ethylene oxide, it was thought advisable to construct a

bag with thicker and tighter walls. To this end, the B-29 inner tube chamber previously described was designed.

B-29 inner tube. Increasing the ethylene oxide concentration will decrease the time required for killing certain micro-organisms *(10)*. The ethylene oxide content of the carboxide gas used was determined by the method of Phillips and Kaye *(8)* and found to be 184 mg. per liter.

For the initial test of the B-29 inner tube chamber, the toes were removed from 6 well-worn shoes and both portions of the shoes were placed in the chamber which was then filled with carboxide to a pressure of 20 cm. of water. One hour after the chamber was filled, the gas content was analyzed and found to contain 34 mg. of ethylene oxide per liter of gas, a loss of over 81 percent of the original content. Three hours later the ethylene oxide concentration had dropped to 31 mg. per liter. Immediately prior to the removal of the shoes, the content of ethylene oxide was found to be 26 mg. per liter. This was an 86 percent loss in 24 hours.

The shoe toes were placed in sterile cellophane bags and aerated for 6 hours. The insides of the toes were then coated with 20 cc. of melted Sabouraud's agar, placed in moist sterile culture dishes, and incubated in a humidified incubator at 37° C. for 6 days. The toes of the mates to the shoes exposed to the gas were treated identically with the treated shoes, except for the exposure to the gas. At the end of a 6-day incubation period the toes of all the control shoes showed luxuriant fungus growth. The treated shoe toes all gave evidence of growth but not such luxuriant growth as that obtained from the control shoes.

Apparently the concentration of ethylene oxide in the gas was not sufficiently high to sterilize the shoes. In the next experiment the ethylene oxide content was increased in the following manner: 90 cc. of liquid ethylene oxide was measured into a vacuum flask which was then stopped and the side arm connected to the valve of the chamber. The vacuum flask was then immersed in warm water and the liquid ethylene oxide vaporized into the chamber. Sufficient carboxide gas was then added to raise the pressure to 20 cm. of water.

Analysis of the gas obtained with this mixture showed it to contain 940 mg. of ethylene oxide per liter of gas. To determine the degree of loss of ethylene oxide through the walls of the chamber, this mixture was allowed to remain in the bag for 24 hours. At that time, analysis of the gas showed it to contain 851 mg. of ethylene oxide per liter, a loss of 10 percent, due probably to a diffusion through the walls of the chamber.

The next experiment consisted of removing the toes from 6 shoes and placing both portions of the shoes in the chamber. Ninety cubic

(10) Kaye, S., and Phillips, C. R.: Sterilizing action of gaseous ethylene oxide; effect of moisture. Am. J. Hyg. 50: 296-306, Nov. 1949.

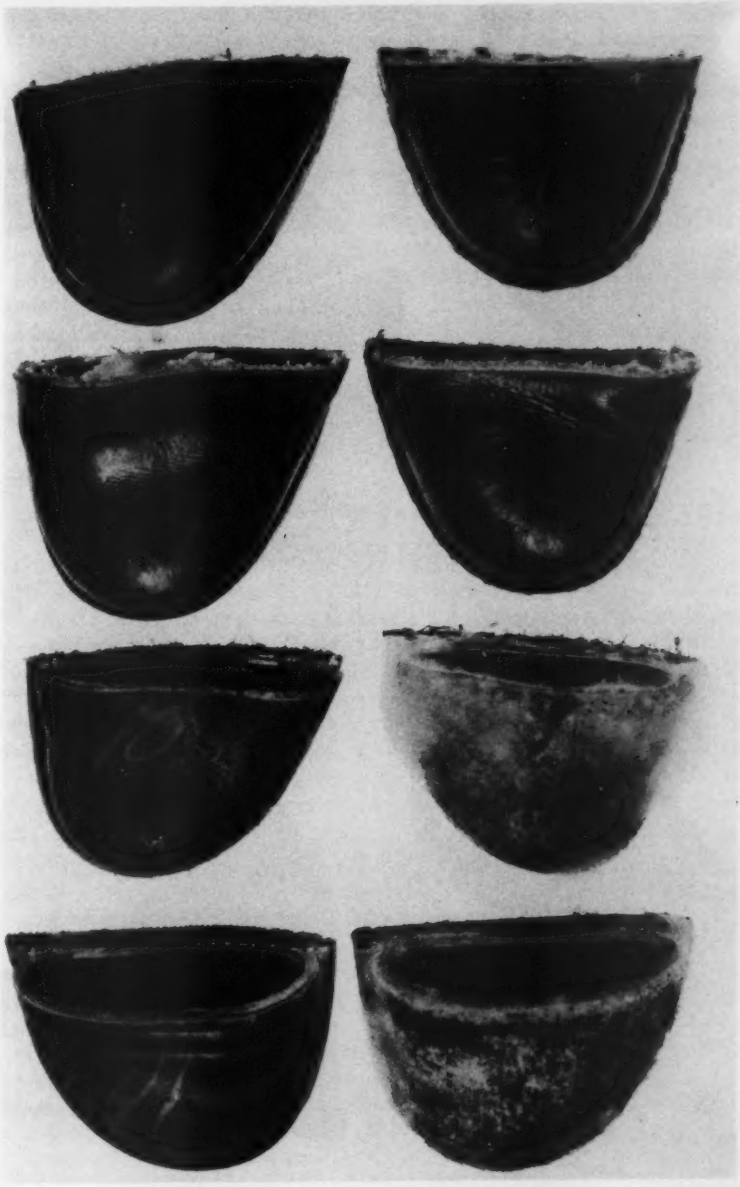

Figure 4. Shoes treated with carboxide gas. Right: Untreated shoes. Left: Treated shoes.

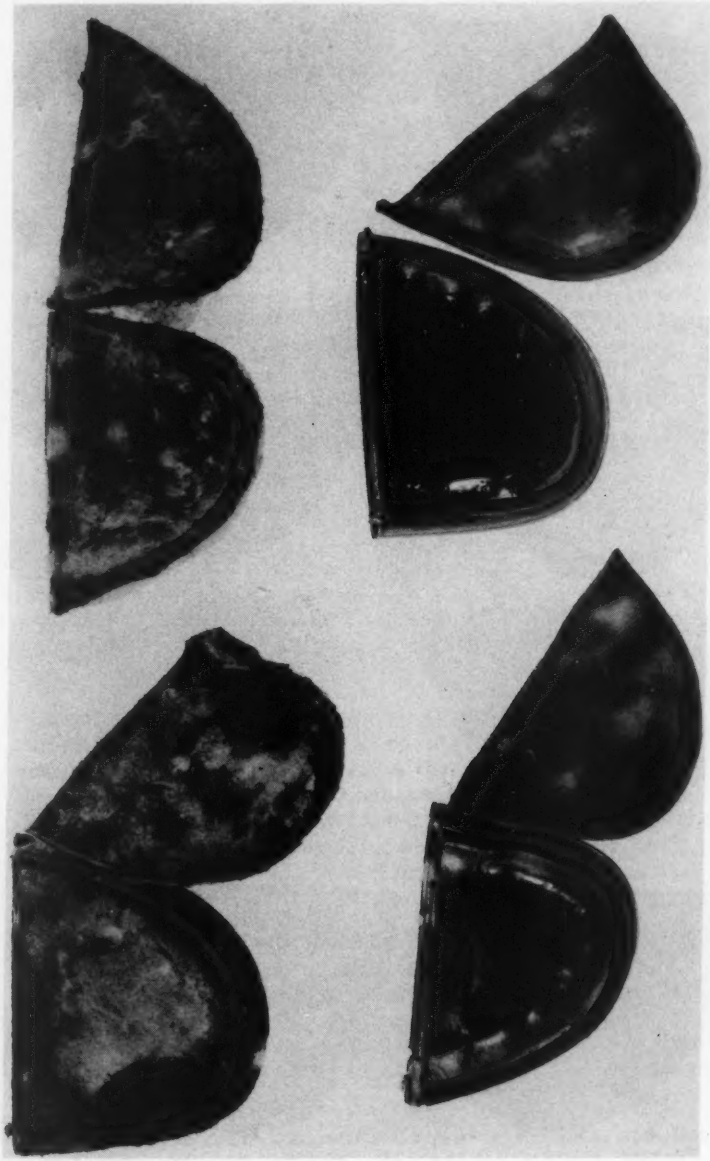

Figure 5. Shoes, Nos. 40 and 41, shown in figure 4 cut open to show the effectiveness of the treatment. Top: Untreated shoes. Bottom: Treated shoes.

centimeters of liquid ethylene oxide was vaporized into the chamber and sufficient carboxide gas was added to raise the pressure to 20 cm. of water. One hour after filling with the gas the contents were analyzed and found to contain 493 mg. of ethylene oxide per liter of gas. This was a loss of about 50 percent from the initial calculated value. Twenty-four hours later, immediately prior to the removal of the shoes, the ethylene oxide content was found to be 320 mg. per liter of gas. As can readily be seen, the loss during the first hour was much greater than that occurring in the following 23 hours. The initial loss may be explained by the diffusion of the ethylene oxide gas into the shoes where it might possibly combine with the moisture in the leather to form ethylene glycol.

After the shoe toes were coated with Sabouraud's agar and incubated for 6 days in a humidified incubator at 37° C., the control or untreated mates to the shoes showed luxuriant growth of fungus, but the treated shoes were completely devoid of growth. Typical results obtained are shown in figures 4 and 5. This experiment was repeated and identical results were obtained.

An additional experiment was accomplished using 45 cc. of liquid ethylene oxide and sufficient carboxide gas to bring the chamber to a pressure of 20 cm. of water. Analysis of this mixture showed the ethylene oxide content to be 520 mg. per liter. Both portions of 4 shoes were placed in the chamber. One hour after filling, analysis of the gas showed the ethylene oxide content to be 225 mg. per liter. Twenty-four hours after initial filling and immediately prior to removal of the shoes the content was found to be 111 mg. of ethylene oxide per liter. The shoe toes were removed, aerated, coated with agar, and incubated by the standard method. At the end of 10 days at 37° C. in the humidified incubator, the control shoes showed abundant fungus growth, whereas no growth was observed on the treated shoes.

A similar experiment was performed but the shoes were exposed for only 12 hours. At the time of removal of the shoes the gas was analyzed and found to contain 154 mg. of ethylene oxide per liter. After 11 days in the humidified incubator at 37° C. all control shoes showed abundant fungus growth, while the treated shoes showed no growth. This experiment was repeated and similar results were obtained.

DISCUSSION

It has been shown that leather samples contaminated with various pathogenic fungi can be sterilized and that the normal nonpathogenic fungi and spores present in well-worn shoes can be destroyed by exposure to certain concentrations of ethylene oxide gas. The leather samples contaminated with pathogenic fungi were sterilized by exposure to a gas containing 184 mg. of ethylene oxide per liter for 24 hours. A higher concentration of ethylene oxide was required to sterilize the whole shoes in the same period of time. This difference was probably

due to the diffusion of the ethylene oxide into the shoes, where it was possibly hydrolyzed into ethylene glycol, thereby reducing the concentration of ethylene oxide available for sterilization. In addition, a certain amount of the ethylene oxide was lost by diffusion through the walls of the exposure bag. This loss, however, was small compared to the gas lost by diffusion into the shoes. Tests have shown that a 10 percent reduction in the concentration of ethylene oxide over a 24-hour period was the maximum loss by diffusion through the walls of the bag. In 24 hours the concentration of ethylene oxide dropped from 940 mg. to 851 mg. per liter of gas. This was equivalent to a loss of 10.6 gm. of liquid ethylene oxide.

When 6 shoes were exposed to an initial concentration of 940 mg. of ethylene oxide per liter of gas in a 120-liter exposure bag there was a 48 percent reduction in the concentration of ethylene oxide within the first hour of exposure. This was equivalent to a loss of 53.5 gm. of liquid ethylene oxide. This would indicate that each shoe had absorbed the equivalent of 8.9 gm. of liquid ethylene oxide in the first hour of exposure. By the end of 24 hours the concentration of ethylene oxide had dropped to 320 mg. per liter of gas. This loss was equivalent to 74.4 gm. of liquid ethylene oxide. If 10.6 gm. were lost because of diffusion through the walls of the exposure bag, then 63.8 gm. of liquid ethylene oxide were taken up by the 6 shoes used in the experiment or 10.6 gm. of liquid ethylene oxide had been absorbed by each shoe.

When 4 shoes were exposed to an initial concentration of 520 mg. of ethylene oxide per liter of gas, the concentration dropped to 225 mg. per liter in the first hour. This was equivalent to a loss of 35.4 gm. of liquid ethylene oxide or 8.8 gm. per shoe. At the end of 24 hours the concentration of ethylene oxide had dropped to 111 mg. per liter. This was equivalent to a loss of 49.6 gm. of liquid ethylene oxide. If 10.6 gm. of liquid ethylene oxide were lost by diffusion through the walls of the bag then each shoe in the experiment absorbed 9.8 gm. of liquid ethylene oxide.

The loss, per shoe exposed, in the 2 experiments was practically the same. This could be expected inasmuch as all of the shoes were about the same size and had been stored under the same conditions for a long time prior to exposure to the gas. It is expected that all of the shoes would have reached the same degree of saturation with moisture during this period of storage and would, therefore, absorb the same quantity of ethylene oxide gas.

Although certain glycols are powerful germicides in aerosol form they exhibit little germicidal action on surfaces or in solutions. For this reason any of the ethylene oxide that was absorbed into the shoe and hydrolyzed to ethylene glycol on combination with the moisture in the shoe would account for a very limited degree of sterilization. The initial concentration of ethylene oxide, therefore, must be sufficiently high to compensate for this initial loss and a sufficient concentration

to accomplish the sterilization must be maintained. The results obtained in this series of experiments would indicate that the ethylene oxide concentration should not drop below 150 mg. per liter of gas throughout a 12-hour exposure period. It may be that much higher initial concentrations of ethylene oxide gas will accomplish the sterilization in much shorter periods of time.

Of the various types of sterilizing chambers constructed and tested the chamber made from the inner tube was by far the most satisfactory. This type of chamber is simple to construct from materials that are readily available under almost any field condition.

Phillips *(11)* has recorded several cases in which severe burns occurred when rubber footwear was worn immediately after treatment and without any aeration. No burns have been recorded when the shoes were aerated after treatment. It is, therefore, necessary to aerate sufficiently all footwear after treatment with ethylene oxide gas.

SUMMARY

Leather samples contaminated with various pathogenic fungi were sterilized by exposure to ethylene oxide gas. Simple exposure chambers to accomplish sterilization of shoes were designed, constructed, and tested. A container improvised from a section of a B-29 type aircraft inner tube was found to be most satisfactory. Well-worn shoes were sterilized by exposure to certain concentrations of ethylene oxide gas.

(11) Phillips, C. R.: Personal communication.

BOOK REVIEWS

Textbook of Refraction, by *Edwin Forbes Tait,* M. D., Ph. D., Associate Professor of Ophthalmology, Temple University School of Medicine; Attending Surgeon (Ophthalmology), Temple University and Montgomery Hospitals; Fellow, Philadelphia College of Physicians, and American Academy of Ophthalmology and Otolaryngology; Member; The Pan-American Association of Ophthalmology, and The Association for Research in Ophthalmology. 418 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1951. Price $8.

This book is far from the textbook of refraction that its title implies. Dr. Tait has, in a comprehensive volume, described his own methods of refraction and beliefs in a philosophic fashion. The basic fundamental knowledge of optics has been quickly dismissed and he has approached the subject from a physiologic viewpoint. Although his chapters on refractive errors are excellent and one can find little quarrel within them, his rather exhaustive treatment of the anomalies and reflexes of the ocular muscles differs from the most accepted ophthalmic theories on muscle balance. The section on refraction is similarly divergent from the accepted methods as practiced by most ophthalmologists and as

described in most textbooks written by ophthalmologists. The use of dynamic skiametry is overemphasized. His evaluation of the use of cycloplegia is at variance with my own experience. I believe that this book has been written primarily for optometrists and although the approach to the subject is new and refreshing I think that this book adds little to our basic knowledge in the practice of refraction in the field of ophthalmology.—*Maj. R. U. Stern, MC, U. S. A.*

Formulary and Therapeutic Guide, by *R. Gordon Douglas,* Chairman, *Donald A. Clarke,* Secretary, and *Norman Baker, Henry Barnett, McKeen Cattell, Herbert Conway, Cary Eggleston, August Groeschel, Vassar Johnson, Vernon Knight,* and *Ade Milhorat.* 355 pages. Appleton-Century-Crofts, Inc., New York, N. Y., publisher, 1951. Price $3.

This book, compiled by the Formulary Committee of the New York Hospital, consists of a preface, alphabetical listing of drugs, equivalent weights and measures, Latin expressions and abbreviations, and a therapeutic index. The preface, of interest primarily to the New York Hospital staff, includes a history of the formulary, information on the formulary system and its implementation, and pharmaceutic procedures and regulations. The articles are comprehensive, both as to content and number, and consist of a description of the drug, usage, dosage, precautions, and how dispensed. Whenever possible, nomenclature follows the U. S. Pharmacopoeia, National Formulary, or New and Nonofficial Remedies. Cross-indexing by synonyms and proprietary names is extensive. The therapeutic index could be more inclusive. No synonyms are listed and page numbers of articles on included drugs are lacking. Although developed for the use of the New York Hospital, other hospitals not having a suitable formulary should find this book of value as it is the best of its type now available.—*Capt. J. W. McNamara, MSC, U. S. A.*

The Pathogenesis of Tuberculosis, by *Arnold R. Rich,* M. D., Bazley Professor of Pathology, The Johns Hopkins University School of Medicine; Pathologist-in-Chief, The Johns Hopkins Hospital, Baltimore. 2d edition. 1,028 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1951. Price $15.

The first edition of this text was published in 1944 and became the best reference text in its field. Not only was it accepted in this country, but the Spanish translation is widely known in South American medical circles. The organization of the material in this edition is the same as that in the first. The excellent bibliography has been expanded to 1,500 references which, in the new edition, includes the pertinent page numbers. I know of no other book which covers in as clear and orderly manner the tremendous amount of work done in the field of experimental tuberculosis. The chapters on hypersensitivity are especially good.—*Lt. Col. J. R. Vivas, MC, U. S. A.*

Review of Tuberculosis Cases, United States Navy, 1949

Epidemiologic Implications *(1)*

Sidney A. Britten, *Commander, MC, U. S. N. (2)*

IN a previous article *(3)* analysis of incidence rates for tuberculosis among naval personnel for the period 1943-1948 exposed differences in respect to sex, age, occupation, and length of service and confirmed the impression that guides were available for the planning of supplementary aids to the more general measures then in effect. Three conclusions were established: (1) attack rates increase with advancing age; (2) the return to duty of personnel with pulmonary defects of undetermined cause necessitates continuous supervision of an increasing group; and (3) a study of individual case records of persons admitted for tuberculosis should be undertaken in an attempt to find answers to questions concerning the higher rates for certain classes of enlisted personnel and for officers of the Navy Nurse Corps. Sources of dissemination of the tubercle bacillus within the service and causes of reduction in host resistance to invasion should be defined and brought under control if possible because about 90 percent of recruits entering the service during 1948-1950 were without evidence of prior infection as determined by negative reactions to intradermal injections of 0.0001 mg. of tuberculin (PPD).

The purpose of this article is to summarize the epidemiologic evidence contained in individual case records of personnel admitted for tuberculosis during 1949, as proposed in the previous study.

The central medical records of all patients tabulated as new cases of tuberculosis for the year *(4)* were reviewed and diagnoses were

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(1) Presented before the Epidemiology Section, American Public Health Association, San Francisco, Calif., 2 November 1951.

(2) Preventive Medicine Division, Bureau of Medicine and Surgery, Department of the Navy.

(3) Britten, S. A., and Charter, W. V.: Focal attack in tuberculosis control. U. S. Armed Forces M. J. 2: 1045-1053, July 1951.

(4) Medical Statistics. Annual Report of the Surgeon General, U. S. Navy, for the Calendar Year 1949. NavMed P-154. p. 38.

assigned which appeared to represent best all the information available. The revised diagnoses are summarized in table 1. Only those cases in which bacteriologic confirmation was obtained, serial roentgenograms revealed evidence of a changing lesion, or pathologic tissue findings were typical are classified "active"; the exceptions to this rule were 2 of the patients with pleural effusion in whom a positive tuberculin skin-reaction was the only evidence of tuberculosis. Of those with active or recently active pulmonary tuberculosis, 29 percent were minimal in extent on admission, 60 percent moderately advanced, and 11 percent far advanced, using the National Tuberculosis Association criteria then in effect (5). Attention had been directed to the pulmonary lesions in 4 patients following their admission to the sick list for extrapulmonary tuberculosis and in 1 patient when a suspected neurofibroma was found to be a quiescent Ghon tubercle following resection. About 25 percent of all the patients counted as new cases of tuberculosis in 1949 had only unchanging roentgenographic evidence of pulmonary defect. Ten patients could be excluded because of subsequent change in diagnosis to nontuberculous conditions or because they had been reported for the first time prior to 1949. In 2 others the evidence completely disappeared and in the remaining patient only a healed primary complex, not considered disqualifying, was the final diagnosis.

As shown in table 2, only 1 of the patients with active or recently active pulmonary tuberculosis and only 1 of those with pleural effusion was returned to duty, the former following successful resection of a Ghon tubercle. Most of those with tuberculous cervical adenitis were returned to duty following surgical and chemotherapeutic treatment, as were about one-third of those with other extrapulmonary forms of active tuberculosis and most of those with unchanging pulmonary or pleural roentgenographic shadows. Although no known communicable cases were retained in the naval service, 122 persons whose future health and epidemiologic importance remain in doubt were returned to duty.

From table 3, it is evident that although the gross annual new case rate for tuberculosis of present clinical significance was comparatively low (only 59 per 100,000 average strength) (6-9) marked variations were observed when specific rates according to type of personnel were considered. The rate for Caucasians was half that for Negroid

(5) Diagnostic Standards and Classification of Tuberculosis. National Tuberculosis Association, New York, N. Y., 1940.

(6) Diagnostic Standards and Classification of Tuberculosis. National Tuberculosis Association, New York, N. Y., 1950. p. 28.

(7) Christie, A. C.: Results of Washington, D. C., chest x-ray survey. Nat. Tuberc. A. Tr. 429, Apr. 1950.

(8) Pierce, R. I.: Symposium on mass chest x-ray surveys; part 2. Nat. Tuberc. A. Tr. 442, Apr. 1950.

(9) Boynton, R. E.: Tuberculosis in American colleges and universities. Dis. Chest 11: 340-350, July-Aug. 1945.

and one-eighth that for all other races. Of the 44 non-Caucasians 43 were Navy enlisted men and 1 was a Marine Corps enlisted man. The relatively low rate for male officers has been consistent through the years *(10, 11)*. The rates for other groups were based on numbers so small that little reliance may be placed on them.

TABLE 1. *Diagnoses (assigned to new tuberculosis cases after case review) and rate per 100,000 average strength for tuberculosis, all forms, United States Navy, 1949*

Diagnoses	Number of cases	Rate per 100,000
Total	442	83
Tuberculosis pulmonary, active, minimal*	75	14
Tuberculosis pulmonary, active, moderately advanced**	166	31
Tuberculosis pulmonary, active, far advanced	29	5
Pleural effusion	10	2
Tuberculous cervical adenitis	15	3
All other active tuberculosis	14	3
Tuberculosis pulmonary, recently active	5	1
Unchanging pulmonary or pleural roentgenographic shadows	115	21
No disease	2	3
Healed primary complex	1	
Sarcoid	1	
Bronchiectasis	1	
Congenital pulmonary cysts	1	
Tuberculous cases of record prior to 1949	7	

*Tuberculous cervical adenitis was diagnosed prior to the pulmonary lesion in 1 patient.

**Pleural effusion, tuberculosis of malleolus, or tuberculous cervical adenitis was diagnosed prior to pulmonary lesion in 3 patients.

Thirty patients were from the Medical Department (120 per 100,000 average strength). The rates for Nurse Corps, Medical Corps, and Hospital Corps deviated only slightly from that of the Medical Department as a whole. Although such factors as selection and exposure before and after entering the service no doubt affected the rates in these groups *(12-14)*, larger numbers are needed for analysis than are

(10) Smiley, D. F., and Raskin, H. A.: Tuberculosis as Navy problem. Dis. Chest 10: 210-233, May-June 1944.

(11) Britten, S. A., and Charter, W. V.: Routine photofluorographic examinations of Naval and Marine Corps personnel; end results. U. S. Nav. M. Bull. 47: 733-738, July-Aug. 1947.

(12) Amberson, J. B., Jr., and Riggins, H. M.: Tuberculosis among student nurses; 5-year study at Belleview Hospital. Ann. Int. Med. 10: 156-165, Aug. 1936.

(13) Bunn, P. A., and Lansdown, F. S.: Observations on tuberculosis control. Ann. Int. Med. 24: 968, June 1946.

(14) Long, E. R.: Hazard of acquiring tuberculosis in laboratory. Am. J. Pub. Health 41: 782-787, July 1951.

TABLE 2. *Disposition of patients following clinical study and treatment for tuberculosis, all forms, United States Navy, 1949*

Disposition	Total[1]	Active or recently active pulmonary tuberculosis	Pleural effusion	Tuberculosis cervical adenitis	All other active tuberculosis	Unchanging pulmonary or pleural shadows in roentgenograms
Total	429	275	10	15	14	115
Invalided from service	286	262	8[4]	1	9	6
Discharge from service	9	4[2]				5
To fleet reserve	3			1[2]		2
Administrative discharge	3	3				
Died	5	4	1[5]			
To duty under observation	21			3	1	17
To duty	101	1[3]	1	10	4[6]	85
Continued under treatment	1	1				

(1) Does not include 13 patients who were eliminated from the study.
(2) Following successful treatment.
(3) Following resection of quiescent Ghon tubercle.
(4) One with residual pulmonary shadows, 1 with advanced pulmonary disease following decortication, 1 following empyema and pneumothorax, and 2 with negative roentgenograms of the chest.
(5) Tuberculous meningitis following recovery from pleural effusion.
(6) Two abscesses, 1 scleritis, and 1 hilar lymphadenitis.

TABLE 3. *Type of personnel, number of patients and rate per 100,000 average strength for tuberculosis all forms, active or recently active, United States Navy, 1949*

Personnel	Number of patients	Rate per 100,000	Personnel	Number of patients	Rate per 100,000
Total	314	59	Status		
Race*			Officers, men	18	33
Caucasian	268	53	Officers, women***	4	160
Negroid	18	100	Enlisted men	286	61
All others**	25	400	Midshipmen (Academy)	3	90
			Prisoners (g. c. m.)	3	100

*Excludes 3 general court martial prisoners.
**Includes Philippine, Chinese, Samoan, Chamorro, and American Indian personnel.
***Includes 3 officers of the Navy Nurse Corps.

available in any 1 year. A 10-year study of tuberculosis among members of the Navy Nurse Corps has been initiated. This may define some of the factors producing the higher rates. Only enlisted men presented a sufficiently large population to warrant analysis of variations within an occupational group, and even there only large differences are statistically significant.

TABLF 4. *Age and years of service, number of patients and rate per 100,000 average strength for tuberculosis, all forms, active or recently active, in enlisted men of the United States Navy, 1949*

Age (years)	Number of patients *(1)*	Rate per 100,000	Years of service	Number of patients *(1, 2)*	Rate per 100,000
Total	286	61	Total	286	61
Under 20	63	50	0–2	138	59
20–24	124	61	3–5	65	62
25–29	49	65	6–8	37	61
30–34	28	75	9–11	14	59
35–39	15	94	12–14	15	100
40 and over	7	96	15–17	4	70
			18–20	6	100
			21 and over	7	220

(1) General court-martial prisoners were not included.
(2) Patients with broken service were tabulated as continuous service if the break in service was less than 1 year. If the break was longer, only the latest enlistment was counted.

The distribution of enlisted men by age and length of service is shown in table 4. From the table it is seen that new case rates increased with increasing age and also with increasing years of service except for the low peak in the early years of service which is apparently explained, at least in part, by the presence of cases of the older age groups with short length of service. So high a proportion of the patients was discovered in the early years of service that the question of

TABLE 5. *Characteristics of cases according to type of personnel for tuberculosis, active and recently active in enlisted men* of the United States Navy, 1949*

Characteristics	Total	Aviation	Other	Medical and dental	Steward
All forms, active or recently active					
Number of patients	286	22	209	23	32
Rate per 100,000 average strength	61	31	58	130	210
Mean age, last birthday (years)	24	27	24	20	26
Mean years of service, last full year	4	6	4	2	6
Non-Caucasian (percent)	15	–	5	–	100
Percent with contact within 5 years	12	13	10	45	3
Patients with nonpulmonary tuberculosis (percent)	14	18	11	13	31
Pulmonary, active or recently active					
Number of patients	248	19	187	20	22
Percent discovered by routine roentgenogram**	50	50	51	40	41
Percent with prior evidence of lesion	30	38	31	30	23
Percent with minimal lesions	25	38	19	45	41

*General court-martial prisoners not included.

**Personnel receive a routine roentgenographic examination of the chest when entering the service, at annual intervals while on active duty, and when leaving the service.

whether or not their disease was more than coincidentally related to naval service should be raised and the answer sought. Routine tuberculin-testing of recruits was begun in 1948; 1949 is too early to relate the results to the later incidence of disease. Of the 23 patients in whom the result of the test is known, 13 were positive and 10 negative; of those whose test was positive there were only 2 in whom the photofluorogram of the chest taken on induction suggested the possibility of tuberculosis. Future studies should furnish information of interest in this regard.

Among the various ratings of enlisted men, such as engineering and hull, deck and ordnance, Marine Corps, seamen, electronics, radio, administration and clerical, recruits, firemen, aviation, stewards, and miscellaneous, the rates for only 3 deviated significantly from that

for all enlisted men. The rate for aviation enlisted was lower; that for medical and dental, and for stewards, higher. In table 5 these groups are arranged in increasing order of rates and various factors are listed, some of which show rather marked differences among the groups. The patients among aviation personnel were older and of longer service than the average. Unfortunately, age and length of service distributions of all aviation personnel were not available. Although a low rate had also been characteristic of this group over the previous 6 years, there is no ready explanation for it. Study of this phenomenon should be pursued.

TABLE 6. *Age and rating, number of patients and rate per 100,000 for tuberculosis all forms, active or recently active, in medical and dental enlisted men of the United States Navy, 1949**

Age	Number of patients	Rate per 100,000	Rating**	Number of patients	Rate per 100,000
Total	23	130	Total	23	130
Under 20	10	160	HA and HN	11	140
20-24	11***	190	HM3 and HM2	11***	290
25-29	2	57			
30 and over	0	-	HM1 and HMC	1	16

*In the absence of average strength records, a count as of 19 August 1949 was used as population data in determining rates.

**Ratings are listed in increasing order of seniority.

***One laboratory infection of the finger.

The steward group, most of whom were non-Caucasian, had the highest rate, which was not unexpected in view of the relatively high rates for non-Caucasians in and out of the service *(15-19)*. The patients were older and of longer service than the average. The percent with nonpulmonary tuberculosis was higher than for other enlisted men. The high proportion of minimal lesions is a tribute to case-finding technics which are so important in this high incidence group.

As seen in table 5, the patients among medical and dental enlisted personnel were younger than the average, had a shorter length of service, gave a history of recent contact more frequently, and had a higher

(15) DeLien, H., and Dahlstrom, A. W.: Ethnic reservoir of tuberculosis. Am. J. Pub. Health 41: 528-532, May 1951.

(16) Stewart, H. C.; Gass, R. S.; Puffer, R. R.; and Williams, W. C.: Tuberculosis studies in Tennessee; morbidity and mortality in white households during period of observation. Am. J. Hyg. 37: 193-205, Mar. 1943.

(17) Young, L. K.: Tuberculosis control in the Philippines. Pub. Health Rep. 64: 123, Feb. 1949.

(18) Tao, J. C.: Pulmonary tuberculosis in Chinese students. Am. Rev. Tuberc. 56: 22-26, July 1947.

(19) Payne, H. M.: Problem of tuberculosis control among American Negroes. Am. Rev. Tuberc. 60: 332-342, Sept. 1949.

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proportion of minimal lesions. Although detailed average strength data were not more available for these than for stewards and aviation enlisted men, a count according to age and rating as of 19 August 1949 was available. Using these data the rates shown in table 6 were computed. There was a very low incidence of new patients among the older medical and dental enlisted personnel. A possible association of the patients with youth and with the ratings most intimately involved in the care of the sick seems to be suggested. The factor of association with care of the sick is not weakened by the fact that contact with tuberculous patients or their discharges within 5 years of admission was known to have occurred in 35 percent of the patients in this group. Contact with tuberculous friends or relatives outside the service was known in 13 percent of the patients, practically the same as that recorded in table 5 for other enlisted personnel among whom known contact in the service was rare. The degree of involvement on admission was minimal in 9 of the 20 patients with pulmonary tuberculosis. The result of a tuberculin test at time of entering the service, and of repeated tests thereafter, would assist greatly in evaluating the association of these patients with care of the sick. Because the Hospital Corps is made up of volunteers, and because the prior experience of applicants is considered when assigning them, the factor of selection as a force producing the higher rates should be considered in future studies.

Past attempts to relate the onset of disease to station of duty have been fruitless because so often the evidence of illness which had its onset elsewhere is not noted until return to the United States where roentgenographic facilities are more generally available. Admissions for illness acquired overseas or in ships may take place after arrival at continental receiving stations and, therefore, be included in the statistics for the continental United States. So, in arriving at the distribution shown in table 7, the station of duty at onset was arbitrarily defined as the last area or ship assignment of more than 2 months' duration. Because the time spent in the area or ship varied from several months to several years, no confidence may be placed in small differences in rates. The exception to a fairly uniform distribution is ships, where the rate for pulmonary disease among patients with no known prior evidence of disease was higher than would have been predicted from experience ashore in the United States or overseas. Should this exception be noted consistently, it would justify the traditional fear of tuberculosis as a marine disease and emphasize the hazard healthy persons face when living and working in close company with a chronic carrier of the tubercle bacillus. The ratio of Caucasian to non-Caucasian patients was about 10:1 for ships and ashore in the United States and 4:1 for ashore, overseas.

On the basis of table 8 tuberculosis should have been discovered in 27 percent of the patients prior to the time that it was, the delay being due to technically unsatisfactory films, failures in interpreting films, and recommendations made improperly or not carried out. The problems

TABLE 7. *Method of discovery and place at onset*, related to prior evidence of lesion, number of patients and rate per 100,000 average strength for pulmonary tuberculosis, active or recently active, United States Navy, 1949*

Prior evidence of lesion in review of records**	Method of discovery	Place at onset						
		Ashore				Ships		Not known
		United States		Overseas				
		Number of patients	Rate per 100,000	Number of patients	Rate per 100,000	Number of patients	Rate per 100,000	Number of patients
Absent	Total	97	31	12	22	78	44	3
	Routine roentgenogram***	49	16	6	11	41	23	1
	Symptoms	48	15	6	11	37	21	2
Present	Total	42	14	9	17	32	18	
	Routine roentgenogram***	25	8	4	8	18	10	
	Symptoms	17	6	5	9	14	8	
Not known	Total							2

*Last ship or station assignment of more than 2 months' duration prior to admission.
**All records and available miniature films were reviewed for evidence that questionable lesions antedated admission.
***Routine roentgenograms made in accordance with directives or as part of the clinical investigation of a nonrespiratory illness.

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of incomplete participation in mass roentgenographic surveys and of human failures in interpreting films and failure to follow up patients continue to challenge the ingenuity of those using that valuable method of case-finding *(20-22)*.

TABLE 8. *Roentgenographic evidence in the record, pulmonary tuberculosis, active or recently active, United States Navy, 1949*

Roentgenographic evidence in the record	Number of patients	Percent
Total available records	272	100
Last prior film negative at review	69	25
Last prior film reported negative but not reviewed*	89	33
Prior film reported negative but questionable on review	38	14
Prior film reported questionable; clinical study; to duty	33	12
No technically satisfactory film in the record	26	10
Recruit film cause for admission**	5	2
Serial films made; progressed under observation	4	1
Prior film reported as cause for clinical study; not made	4	1
Disqualifying lesion described; clinical study not recommended	3	1
Lesion noted at time of discharge from prior enlistment	1	-
Record not available or incomplete***	3	

*Thirty-four records contained films of earlier date which were negative at review.
**Two marine recruits and 3 naval recruits.
***These are not included in the total.

Another matter giving concern to those involved in mass chest surveys are the patients with no evidence of tuberculosis other than a positive tuberculin test and unchanging roentgenographic findings *(23,24)*. Twelve percent of the patients with active or recently active pulmonary tuberculosis in this study had been hospitalized previously for questionable roentgenographic findings and returned to duty with various nonspecific diagnoses after clinical and laboratory studies. Be-

(20) Burke, M. H.; Schenck, H. C.; and Thrash, J. A.: Tuberculosis studies in Muscogee County, Ga.; x-ray findings in community-wide survey and its coverage as determined by population census. Pub. Health Rep. 64: 263-290, Mar. 4, 1949.

(21) Silverman, C.: Appraisal of contribution of mass radiography in discovery of pulmonary tuberculosis. Am. Rev. Tuberc. 60: 466-482, Oct. 1949.

(22) Zwerling, H. B.; Miller, E. R.; Harkness, J. T.; and Yerushalmy, J.: Clinical importance of lesions undetected in mass radiographic survey of chest. Am. Rev. Tuberc. 64: 249-255, Sept. 1951.

(23) Reisner, D., and Rikli, A.: Community-wide chest x-ray survey. Pub. Health Rep. 66: 423-443, Apr. 6, 1951.

(24) Northrup, C.; Anderson, R. J.; and Sauer, H. I.: What is reportable case of tuberculosis? Pub. Health Rep. 64: 961-968, Aug. 5, 1949.

cause most of these patients had positive sputum and chronic cough, they were probable public health hazards at the time the true nature of their illness was discovered. In many instances, their pulmonary involvement had greatly increased in the interval between their return to duty and the establishment of the diagnosis.

TABLE 9. *Months between admission to sick list and last prior negative routine roentgenographic examination of the chest* for pulmonary tuberculosis, active or recently active, United States Navy, 1949*

Months	Number of patients**	Percent
Total	69	100
0-2***	2	
3-5	4	25
6-8	11	
9-11	19	
12-14	9	41
15-17	8	
18-20	5	
21-23	1	
24-35	5	34
36-47	4	
48 and over	1	

* Includes only patients whose last prior film was negative at review.
** Recruits not included.
*** Shortest interval was 7 days.

The limitations of any method of case-finding should be known. Hence, analysis of the records of the 158 patients whose last prior film was reported negative would be of interest from the standpoint of time lapse between that examination and the admission for illness. Because it was believed that only those cases should be tabulated in which the film in question was available for review and reinterpreted as negative, consideration was limited to 69 patients (table 9). If their experience is any indication of the experience of the rest, then at least 25 percent of the patients whose last prior routine chest roentgenogram was negative became ill before their next annual examination would ordinarily have been scheduled, and 34 percent had missed one or more annual examinations. It is important not only that annual roentgenographic examinations be made promptly, but also that careful investigation of persons presenting themselves at sick call with symptoms be not neglected even though the interval since the last normal chest film may have been only a few days. Not only must the possibility of an acute onset of tuberculosis be borne in mind, but also the possibility that active pulmonary disease may have been present when the roentgenogram was reported negative (being obscured by the shadow of the heart, mediastinum, or diaphragm). Of the 17 patients listed in table 9 as having an interval of less than 9 months between admission and last prior negative chest film, all had symptoms and 7 had physical signs of pulmonary disease;

8 had sputum smears positive for acid-fast bacilli, and 7 more had positive sputum or gastric lavage cultures. Five patients were admitted because of hemoptysis, and 1 each for what was thought to be acute mononucleosis, influenza, primary atypical pneumonia, and pneumonia of unspecified nature treated with penicillin. Persistent cough, fever, and loss of weight were frequent symptoms. Investigation of chest pain in 2 patients and of a unilateral "wheeze" in 1 resulted in establishing the true diagnosis.

SUMMARY AND CONCLUSIONS

The principal epidemiologic findings in this review corroborate those of previous reports. Although the new case rate for tuberculosis, all forms, active or recently active, for 1949 was low (59 per 100,000 average strength), marked variations were noted when rates for specific types of personnel were considered. Highest rates occurred among the non-Caucasian races, particularly those of oriental and Malayan origin. By occupation, stewards had the highest rate, in keeping with their racial composition, and Medical Department personnel had the next highest. The rates for the skilled craftsmen were widely scattered above the mean; those for seamen, marines, aviation personnel, and male officers were below the mean.

The explanation for the low rates for male officers and aviation enlisted men is not readily apparent, nor is that for the higher rates among the non-Caucasians, who received the same physical examinations and roentgenographic screening as other naval personnel.

Although an association between the cases developing among Medical Department personnel and the care of patients was suggested, the number of patients was too small for detailed analysis of this and other factors. In the hope of assessing such factors, a 10-year study of tuberculosis among members of the Navy Nurse Corps was initiated.

The difficulty in identifying sources of infection, even in such broad categories as service on ships or foreign stations, or hospitals, has again been demonstrated and attests to the importance of periodic retesting with tuberculin of naval personnel after they have left the training stations for widely scattered parts of the globe. In order to attack this problem periodic tuberculin-testing and x-raying of Medical Department personnel on duty at naval hospitals was begun in November 1951. This measure may (1) assist in locating sources of infection, (2) identify the persons whose recent infection should be cause for close observation and other precautionary measures, and (3) serve as a pattern for wider use of such case-finding methods in the Navy.

Because from 10 to 15 percent of the patients had had known contact with tuberculosis outside the service, the importance of the home as a source of infection of naval personnel is recognized. Beginning in July 1951, the dependents, 15 years of age or older, of naval personnel have

been encouraged to have routine roentgenographic examinations of the chest at the same places and intervals as naval personnel, the examinations to be made by the Navy when practicable.

Technically unsatisfactory photofluorographic films of the chest, or films reported negative but found to be positive on review were part of the records of over 20 percent of the patients with active or recently active pulmonary tuberculosis. Beginning in June 1951, the photofluorograms of all recruits whose tuberculin tests are positive or doubtful have been interpreted a second time. A second reading may be expected to discover an additional 10 percent of questionable shadows as shown by experience in the Navy and elsewhere.

The importance of prompt scheduling of personnel for required annual chest roentgenographic examinations, and of careful clinical investigation of persons complaining of symptoms even a few days or weeks after such an examination, is again emphasized.

BOOK REVIEWS

Studies in Medicine, A Volume of Papers in Honor of Robert Wood Keeton, by 57 contributors. Foreword by *A. C. Ivy*, Ph. D., M. D. 396 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1951. Price $8.50.

This book was prepared in honor of Robert W. Keeton and was presented to him on the occasion of his retirement as chairman of the Department of Medicine at the University of Illinois. It consists of contributions from many of his friends, associates, and students and reflects his diversified interests. The subjects covered embrace such diverse problems as overnutrition, nutrition in surgery, the management of diabetes, the role of protein in liver disease, diagnostic bone marrow patterns, the psychiatric implication of dental erosion, the functional disturbances resulting from man's upright posture, and the art of using lantern slides. The contributors are distinguished in their respective fields. The book has more than passing value and represents significant research in the broad field of medicine.

—Capt. C. L. Andrews, MC, U. S. N.

Aphorisms of Dr. Charles Horace Mayo, 1865-1939, and Dr. William James Mayo, 1861-1939, collected by *Fredrick A. Willius*, M. D. 109 pages. Charles C Thomas, Publisher, Springfield, Ill., 1951.

Dr. Willius undertook to select aphorisms from the 988 written works of the Mayo brothers and in so doing he became impressed with the value of making the thoughts of these great scientists and humanitarians available to those who had not been privileged to know them.

This small volume is the result. One hundred and seven of the sayings of "Dr. Charlie" are quoted, ranging through the fields of surgical practice, medical education, philosophy of life and political geography. The sources of these sayings are given in a bibliography. "Dr. Will" is quoted in 108 aphorisms ranging through the same general fields but differing in the distinct impress of his own personality. The appendix contains the so-called "Lost Oration" by "Dr. Will" and his letter regarding a gift to the University of Minnesota. In 1934 "Dr. Charlie" made this prophetic statement: "Long ago I learned from my father to put old people to bed only for as short a time as was absolutely necessary, for they were like a foundered horse, if they got down it was difficult for them to get up, and their strength ebbed away very rapidly while in bed." "Dr. Will" is credited with this gem in 1938: "Surgery is more a matter of mental grasp than it is of handicraftsmanship." This should be etched on the mind of every resident in surgery. This is a splendid little volume which should be in the hands of students of all ages.—*Col. W. F. Bowers, MC, U. S. A.*

Atlas of Genito-Urinary Surgery, by *Philip R. Roen,* M. D., F. A. C. S., Instructor in Urology, New York Post-Graduate Medical School; Clinical Instructor in Urology, New York Medical College; Associate Visiting Urologist, Morrisania City Hospital; Assistant Attending Urologist at Midtown Hospital and Yonkers General Hospital; Civilian Consultant in Urology, U. S. Air Force, Mitchel Field Air Base Station Hospital; Diplomate, American Board of Urology. Introduction by *Clarence G. Bandler,* M. D., F. A. C. S., Illustrations by *Charles Stern.* 325 pages; illustrated. Appleton-Century-Crofts, Inc., New York, N. Y., publisher, 1951.

Dr. Roen has presented a long-needed book of illustrative operative urology. This work, while primarily published as a visual aid to students in urology, can be well used by the general surgeon or the trained urologist. Before the publication of this book no modern reference covering the entire operative field of urology with descriptive illustrations was available in one volume. The descriptions are brief and in large bold print. The operative illustrations are placed on the opposing page for easy reference. The simple line drawings illustrate step-by-step progression. The essentials of good urologic surgery are presented. New technics such as retropubic prostatectomy are included. A casual review would lead to the impression that many subjects are too briefly covered, but further study reveals that the author has not repeated procedures covered under other headings. Some improvements will undoubtedly be made in subsequent editions. Further illustrations and descriptions of technically complicated subjects such as transurethral resection of the prostate would be of great value. Dr. Roen has illustrated in some cases more than one acceptable operative technic. It is believed, however, that he has not included the most accepted procedure dealing with some operations.

—*Col. J. A. Schindler, U. S. A. F. (MC)*

Duodenal Ulcer in Military Personnel: Studies on Military Effectiveness of the Ulcer Patient

I. The Problem

Eddy D. Palmer, *Lieutenant Colonel, MC, U. S. A.* (1)

B. H. Sullivan, Jr., *Lieutenant Colonel, MC, U. S. A.* (2)

DUODENAL ulcer constitutes one of the major medical causes of manpower loss by the Armed Forces. In a 2-year period for the Army and a 3-year period for the Navy during World War II, 77,000 men were affected and over 48,000 were discharged from the service because of this disease (3). It has recently been estimated that in this country as many as 10 percent of the adult male population have or will have duodenal ulcer (4). Such a high incidence in the civil population constitutes a loss to the Armed Forces of a large segment of the otherwise available manpower. When duodenal ulcer is diagnosed in active-duty personnel, service regulations specify that disposition must be made according to the professional judgment of the medical officers responsible for each case. Individual officers have developed differing attitudes concerning the value of the ulcer patient to the service, and the influence of service life on the patient and his disease. Thus, at one Army general hospital the concerted professional opinion of the surgical and medical staffs has been that almost every ulcer patient can and should be returned to duty. The patient who cannot be adequately controlled by the usual medical means, they believe, can be rendered

(1) Walter Reed Army Hospital, Washington, D. C.

(2) Brooke Army Hospital, Fort Sam Houston, Tex.

(3) Sandweiss, D. J.: Present status of investigations on peptic ulcer; report of survey by National Committee of American Gastroenterological Association for Study of Peptic Ulcer. Gastroenterology 9: 335-356, Oct. 1947.

(4) Ivy, A. C.; Grossman, M. I.; and Bachrach, W. H.: Peptic Ulcer. The Blakiston Co., Philadelphia, Pa., 1950. p. 608.

fit for general service (duty without restrictions) by appropriate surgical measures. In another general hospital about half of the patients with duodenal ulcer have recently been declared unfit for duty at the completion of the hospitalization period during which the diagnosis was established. Other medical officers have been adamant in their insistence that no ulcer patient should remain in the military service.

As in civil life, many servicemen with ulcers are intense, hard-driving persons who perform satisfactorily in positions of responsibility. Unit commanders find these desirable attributes, and frequently request reassignment of the soldier to his original unit at the completion of his hospitalization. The military records of officers are apt to be proportionately better than those of enlisted men with ulcers. Just as the financial responsibilities and pressures felt by the successful business executive appear to be associated causally with his ulcer, so do the paternal and military responsibilities of the efficient officer render duodenal ulcer a distinct occupational hazard. One would not care to go further and to suggest that the efficiency of a group of officers might be gaged by its ulcer rate, but at least history teaches that a great many of the military geniuses of the world have suffered continuously with, and sometimes died from, peptic ulcer. There appears to be little middle ground: the soldier with ulcer who does not prove superior is usually notably inferior in his own performance and in his influence on his unit's effectiveness. This is one explanation for the attempt of some medical officers to individualize disposition on non- or quasi-medical grounds.

A medical officer's note on an ulcer patient's transfer chart recently received at a general hospital contained an excellent exposition of the middle-of-the-road attitude:

> "In my opinion a soldier with peptic ulcer who has less than 6 years' service has no place in the Regular Army, which should be a hard core around which a civilian army is built. This belief is based on the experience that during the hardships of training and combat, persons with peptic ulcer will probably become liabilities in that (1) many will use their ulcer history as a means of getting out of undesirable or hazardous duties; and (2) the psychic and physical requirements of training and combat contribute largely to the recurrence of ulcers. The result is loss of a key person just when he is most needed and can least be spared, additional work for already overworked medical and administrative personnel, and one more man to be sent back to the zone of the interior.
>
> "I believe that when a man has more service, especially if he has more than 12 years, 2 other factors should be considered. First, the moral responsibility we have toward a man who has given 12 or more years of his life to the military service; and second, the chance that the increased length of service has given him much technical knowledge, which may outweigh the hazard of retaining him in the military service in spite of his ulcer.

"When a man who has between 6 and 12 years service is discovered to have a peptic ulcer I think that the decision concerning his retention in the service should be based on (1) his desire to remain (for if he does not want to remain, he will complain of ulcer symptoms whenever it is advantageous for him to do so); (2) our opinion of his psychic stability; and (3) our opinion of his potential value to the service." (5).

The many points for consideration in the foregoing statement do not bear directly on the essential item, i. e., the probability of recurrence of the ulcer. If the decision regarding qualification for service is based on the individual patient's length of service, proficiency, level of training, or military specialty, the matter becomes administrative rather than medical. A policy based on such considerations might result in salvage of certain key personnel, but it would be ineffective as a general device for retention of the best soldiers and officers, because the information available to the physician ordinarily would not permit judgment of the matter. The unfairness of individual nonmedical criteria for medically terminating an Army career or of permitting desired release from the service, possibly with financial compensation, is apparent. The medical condition becomes sublimated to administrative factors to the extent that the prognosis of the disease is eliminated from consideration.

A practical note dictates that disposition of the ulcer patient may well vary with the type of army which is being maintained at the moment. Thus, when the size of the Army is limited by Congress to a few hundred thousand men, it may be that the best interests of the organization are served by separating the ulcer patient, whatever may be his abilities, in exchange for a healthy recruit. In a national emergency, on the other hand, such a procedure could not be considered, for the Army would require the services of both men. In the latter situation, the limited-duty category preserves the strength of the organization, and makes a valid substitution of individualization of the disease process as a whole for individualization of the patient himself.

Detailed reports evaluating the military effectiveness of the Nazi "ulcer battalions" during World War II would be helpful. If soldiers with ulcers are good soldiers, do they mainatin their effectiveness when brought together in special units, and are the medical and administrative problems incident to the disease simplified? Or does official recognition of unit disability in the end render the unit ineffective as a whole? We know nothing of the "ulcer battalions" but it has been possible to obtain some general impressions from certain Austrian physicians who were associated with this military experiment. (6). The "experiment" was dictated by military necessity in that the services of the

(5) Hoagland, R. J., Colonel, MC, U. S. A.
(6) Schmid, J.: Personal communication.

soldier with ulcer had to be used. The unique feature was the segregation of officers and soldiers into units composed entirely of persons with ulcer. Although at first it had been hoped that the "ulcer battalions" could be assigned exclusively to noncombatant duties, toward the end of the war it became necessary to use them in the line. Then, when possible, a 3-month rotation system was used between combat and noncombat assignments, similar to the system used for German medical students during the war. A 500-bed "stomach hospital" was maintained in the vicinity of Vienna for the care of the "ulcer battalions," when the tactical situation permitted, and for upper gastrointestinal tract problems in general. Although no official opinion on this system is at the moment available, it is known that the physicians concerned came to believe that not only was the administrative problem of proper organization and use of ulcer units so difficult as to be fantastic, but also the personalities and complaints of the individuals (not their actual illnesses) were such that the expected degree of invalidism of the units as a whole was multiplied many times. In the final desperate military situation, when the whole male population had to join in the fighting, the ulcer units were conspicuous for their purported physical indisposition. There was the tendency to make the most of their officially recognized disability. The individual soldier had so long been categorized as a half-soldier, with the privileges incident thereto, that he could not understand why very much should be expected of him and his unit in a crisis.

There are 2 distinct aspects to the problem posed by the ulcer patient who is returned to duty, that of the effect on the patient's health and that of the effect on the efficiency of the Army. The former cannot be evaluated at the moment. The argument may be voiced that the Army is not a social-service organization, and that, even though the ulcer patient may remain healthier in the Army than out, the effectiveness of the Army must be the only consideration. Governed to a large extent by the patient's motivations, it is, nevertheless, a common observation that the quality and regularity of the meals provided in the Army are in some instances of more subjective and objective benefit to the ulcer patient than are those encountered in civil life.

The problem of the potential inductee who is known to have or have had a peptic ulcer properly belongs in this discussion, even though the moral pressures exerted by the eventual public financial obligations to the inductee may be quite different. In some instances the induction of a man with an ulcer would accrue more benefit than liability to the service. Because, it is presumed, this would be true in a minority of the cases and because for induction purposes a uniform policy of physical standards is a practical necessity, the diagnosis of ulcer has been grounds for physical disqualification. It frequently happens, however, that following induction a soldier is discovered to have a duodenal ulcer which in all probability existed prior to service. This occurs often enough that if, in truth, the soldier or officer who has an ulcer con-

stitutes a significant liability to the service, the routine use of more critical gastrointestinal evaluation methods prior to induction would pay important dividends.

In the final analysis it is the ulcer patient's motivation which determines whether he will be a help or a hindrance to the Army. However the problem may be argued, the facts necessitate recognition of motivation as the keystone of proper dispostion. A favorite Shakespearean quotation of the military psychiatrist reads:

"He which hath no stomach to fight
Let him depart; his passport shall be made
And crowns for convoy put into his purse;
We would not die in that man's company
That fears his fellowship to die with us."

For present purposes this can be taken literally, but the disposition problem is not solved thereby, for, if the decision to separate the ulcer patient from the service is made largely on his lack of motivation for duty, the ulcer itself has been given no weight in the matter and one may just as honestly separate on medical grounds the healthy drafted soldier who is indisposed to carry out his assignments. As soon as the presence of an ulcer becomes merely a handy medical excuse for separating an unwanted soldier or officer, a farce is created.

It is personal experience and personal interpretation of the ulcer problem that have governed local customs regarding the disposition of ulcer patients. If an objective study be made to assess the value of soldiers and officers with ulcers to the Armed Forces, it might take the form of a measurement of the noneffective periods due to illness incurred by the ulcer patient who has been returned to duty. Such a study would be only a measure of the negative aspect of the problem. There is no valid method for objective measurement of the positive value of the ulcer patient to the Army.

BOOK REVIEWS

The Specialties in General Practice, edited by *Russell L. Cecil*, M. D., Professor of Clinical Medicine, Emeritus, Cornell University Medical College, New York City, with 21 contributors. 818 pages; illustrated. W. B. Saunders Co., Philadelphia, Pa., publisher, 1951. Price $14.50.

Dr. Cecil has edited a much needed book using outstanding authorities in varied specialties on special subjects. The specialties discussed bear particularly on the everyday problems of the doctors located in isolated areas where early consultation with specialists is not available. The book should assist the general practitioner in diagnosing and treating the many diversified conditions he sees. This

book should also assist him in planning for treatment of his patients or give him information that would lead him to refer the patient to a specialist for further specialized care. In many instances the material discussed is basic but is the very information the practitioner is most likely to need. It is amazing that such a wide coverage of the varied specialties could be condensed into a single volume of this size. It is well illustrated. In short, here is a book written by specialists for the nonspecialist, dealing with the everyday problems of the physician.—*Col. E. M. Smith, MC, U. S. A.*

Scoliosis, Pathology, Etiology, and Treatment, by *Samuel Kleinberg,* M. D., Attending Orthopaedic Surgeon, Hospital for Joint Diseases; Consulting Orthopaedic Surgeon, Lebanon Hospital, Maimonides Hospital, Hospital for Special Surgery, New York City; Lecturer in Orthopaedics, New York University Medical School; Member, American Orthopaedic Association; Member, American Academy of Orthopaedic Surgery; Fellow, American College of Surgeons; Fellow, New York Academy of Medicine; Member, American Academy of Compensation Medicine. 286 pages; illustrated. The Williams & Wilkins Company, Baltimore, Md., publisher, 1951. Price $7.50.

This is a well-written monograph on a subject which is constantly confronting orthopedists but which very few understand thoroughly. Part 1 covers anatomy, pathology, and etiology and gives an excellent background for the basis of treatment. It is well illustrated with photographs and roentgenograms. The chapter on the cause of scoliosis is well outlined and instructive as too often this phase is overlooked by the busy practitioner. The causative role of heredity is emphasized but not thoroughly explained. Part 2 on treatment contains a good chapter on preventive treatment and the various types of treatment available for structural scoliosis are discussed under the headings of gymnastic exercises, supportive apparatus, corrective apparatus, forcible correction, and surgery. In the operative treatment, the use of beef-bone grafts is advocated, a method probably not now popular because of the existence of human bone banks. Apparently in the hands of the author the beef-bone graft has been successful. This monograph is an excellent reference work for the busy orthopedist and includes a well-selected bibliography.

—*Col. A. W. Spittler, MC, U. S. A.*

Treatment of Leukemia in Children[1]

Ogden C. Bruton, *Colonel, MC, U. S. A.*

Walter S. Price, *Major, MC, U. S. A.*

SINCE the original reports of Farber et al. (2) that complete temporary remission occurred in a significant percent of children with acute leukemia following the use of folic acid antagonist drugs, many observers (3-7) have reported an apparent increase in longevity in children responding to these drugs. More recently similar observations have been made following the use of ACTH and cortisone (8-11). Although these materials produce some interesting changes when administered to certain patients with leukemia, most authors think of them as purely experimental drugs because no cure for leukemia is known at this time. Since many fatal conditions are treated with equal vigor and often with less response, we do not believe that such a fatalistic attitude should be applied in treating the leukemic

(1) Walter Reed Army Hospital, Washington, D. C.

(2) Farber, S.; Diamond, L. R.; Mercer, R. D.; Sylvester, R. F.; and Wolff, J. A.: Temporary remissions in acute leukemia in children produced by folic acid antagonist, 4-aminopteroyl-glutamic acid (aminopterin). New England J. Med. 238: 787-793, June 3, 1948.

(3) Dameshek, W.: Use of folic acid antagonists in treatment of acute and subacute leukemia. Blood 4: 168, 1949.

(4) Farber, S.: Some observations on effect of folic acid antagonists on acute leukemia and other forms of incurable cancer. Blood 4: 160, 1949.

(5) Stickney, J. M.; Mills, S. D.; Hagedorn, A. B.; and Cooper, T.: Treatment of acute leukemia with folic acid antagonists. Proc. Staff Meet., Mayo Clin. 24: 525-533, Oct. 12, 1949.

(6) Werber, E. J.; Karpinski, F. E., Jr.; and Heinle, R. W.: Treatment of acute leukemias of children with folic acid antagonists. J. Pediat. 36: 69, 1950.

(7) Rice, C. E.: Personal communication.

(8) Stickney, J. M.; Heck, F. J.; and Watkins, C. H.: Cortisone and ACTH in management of leukemia and lymphoblastoma. Proc. Staff Meet., Mayo Clin. 25: 488-489, Aug. 16, 1950.

(9) Wintrobe, M. M.: ACTH and cortisone in hemopoietic disorders. Am. J. Med. 9: 715-717, Dec. 1950.

(10) Snelling, C. E.; Donohue, W. L.; Laski, B.; and Jackson, S. H.: Pituitary adrenocorticotropic hormone (ACTH) and 11-dehydro-17 hydroxy corticosterone (cortisone) therapy in leukemias and lymphomas of children. Pediatrics 8: 22-25, July 1951.

(11) Schulman, I.; Lanman, J. T.; Laxdal, O. E.; and Holt, L. E., Jr.: Effects of ACTH and cortisone on leukemia in childhood. Pediatrics 8: 34-52, July 1951.

TABLE 1. *Summary of treatment of 15 children with acute leukemia**

Patient	Age (years)	Sex	Aminopterin	ACTH or cortisone	Remission			Time between		Remarks
					Clinical	Peripheral blood	Bone marrow	Onset and treatment (weeks)	Treatment and death (weeks)	
1	5	M	+		−	−	−	11	1/7	
2	2	M	+		−	−	−	2	3	
3	6	F	+		+	+	+	5	68	
4	8	F	+		+	+	+	12	48	Aminopterin only
5	4	M	+		−	−	−	2	2/7	
6	7	M	+		+	−	−	4	32	
7	7/12	F	+		−	−	−	6	1	
8	4	F	+	+	−	−	−	3	3	Aminopterin and
9	5	F	+	+	+	+	+	1	36	ACTH or corti-
10	3	F	+	+	−	−	−	14	1	sone concurrently
11	2½	M		+	+	−	−	16	22	ACTH or corti-
12	4	F	+	+	+	+	+	6	**	sone given prior
13	7	M	+	+	+	+	+	4	25	to admission
14	2	M	+	+	+	−	−	6	26	Aminopterin failed
15	4	M			?+	−	−	4	3	Replacement transfusion given

*All received blood and antibiotics
**Patient was alive and in a remission 58 weeks after treatment was started.
M = male F = female

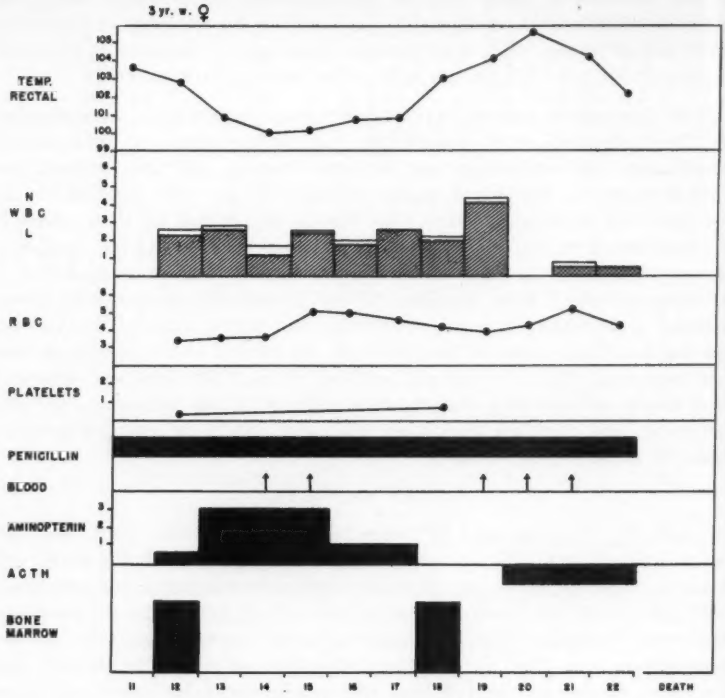
child, but believe rather that all the means at our command should be used to make him as comfortable as possible for as long as possible. This report deals with observations made on 15 children with acute leukemia observed in this hospital in the past 2 years (table 1).

The therapeutic agents used were blood, antibiotics, aminopterin, ACTH, cortisone, and x-ray. One patient received a replacement transfusion and antibiotics as the only therapy. All the patients received blood as needed; 14 received penicillin or other antibiotics; 13 received aminopterin at some time during the course of their therapy; 7 received either ACTH or cortisone at some time during the course of therapy; and 1 who had a destructive lesion with radiculitis in a vertebra received x-ray focally. Of the 14 patients treated with aminopterin, a hormone, or both, 8 (57 percent) had a definite clinical response and 5 (35 percent) had clinical, peripheral blood, and bone marrow response. With this type of combined therapy the response seems to be clearly defined into one of three categories; no response, clinical response, and complete temporary remission including bone marrow. An example of each type follows.

CASE REPORTS

Case 10. A 3-year-old girl was admitted on 11 May 1951. She had been normal until 22 January 1951 when she developed fever and diarrhea. She improved on antibiotic therapy temporarily but was never well afterward and developed an anemia which led to the diagnosis of leukemia 3 months later. On admission she appeared acutely ill. Her temperature was 103° F. and she complained of severe abdominal pain and pain in moving the left leg. Her stools and urine contained gross blood, and blood was seen oozing from the rectum without stool. The spleen was palpable 1 or 2 fingerbreadths below the costal margin and her abdomen was tender throughout. She was moderately anemic. The leukocyte count was 2,550 with 94 percent lymphocytes and 6 percent neutrophils. She was started immediately on antibiotics and following a microscopic examination of the bone marrow, which confirmed the diagnosis, aminopterin therapy was begun. She was given sufficient blood to bring the peripheral blood count to normal, but her course was steadily downhill with increased bleeding from mucous membranes, kidneys, and skin on the fifth day of hospitalization. These symptoms could not be distinguished from aminopterin toxicity so the drug was stopped. Her bone marrow remained unchanged. After 2 days without aminopterin, she was given 100 mg. of ACTH daily for 3 days, during which time she continued her downhill course and died on the eleventh hospital day (fig. 1). This patient is illustrative of those in whom there is no response to treatment.

Case 6. A 7-year-old boy was admitted on 16 November 1950. He had been normal until the first week in September 1950 when he began to have a low grade fever and leg pains. He did not improve on antibiotics and microscopic examination of the bone marrow led to diagnosis of



N (unshaded area) = neutrophils
L (shaded area) = lymphocytes

Figure 1 (case 10). No response to treatment.

acute lymphatic leukemia 1 month later. He received several transfusions and continued to have a low grade fever with bouts of higher fever until his admission to this hospital. Here he appeared pale, but not acutely ill, had no skin lesions, no significant lymphadenopathy, and his liver and spleen were not palpable. His leukocyte count was 2,500 with 12 percent neutrophils and 86 percent lymphocytes. The red blood cells and hemoglobin were normal. He was started on aminopterin. During the first week he ran a febrile course. Penicillin was given and his fever gradually subsided. After 2 weeks he developed troublesome nosebleeds, and after 3 weeks was found to have ulcers on the buccal mucosa. This was interpreted as aminopterin intoxication. He improved rapidly after the aminopterin was discontinued but there was no appreciable change in his peripheral blood count and there was no change in his bone marrow. He was discharged 5 weeks after admission, clinically improved, but without hematologic or bone marrow remission. He is reported to have been fairly well for 2 months, when he was given

another transfusion. He survived 6 months from the date of discharge without further treatment (fig. 2). This patient is typical of those in whom the response to treatment is clinical only.

Case 12. A 4-year-old girl was admitted on 23 February 1951. She had been normal until 30 October 1950 when she developed a high fever and pains in her lower extremities. Hospitalization led to a diagnosis of rheumatic fever in spite of a leukocyte count of 4,000 with 93 percent lymphocytes. During the next 2 months she was given a course of ACTH and 2 courses of cortisone. She responded "dramatically" to the ACTH, less well to the first course of cortisone, and showed no appreciable change to the third course of hormones. On admission she was acutely ill with a temperature of 103° F. She was irritable and complained bitterly of any attempt to move her because of pain in all extremities. Her abdomen was distended and her spleen could not be palpated. The leukocyte count was 2,650 with 97 percent abnormal lymphocytes. She showed moderate anemia and a diminished platelet count. Microscopic examination of the bone marrow confirmed the diagnosis of acute lymphatic leukemia.

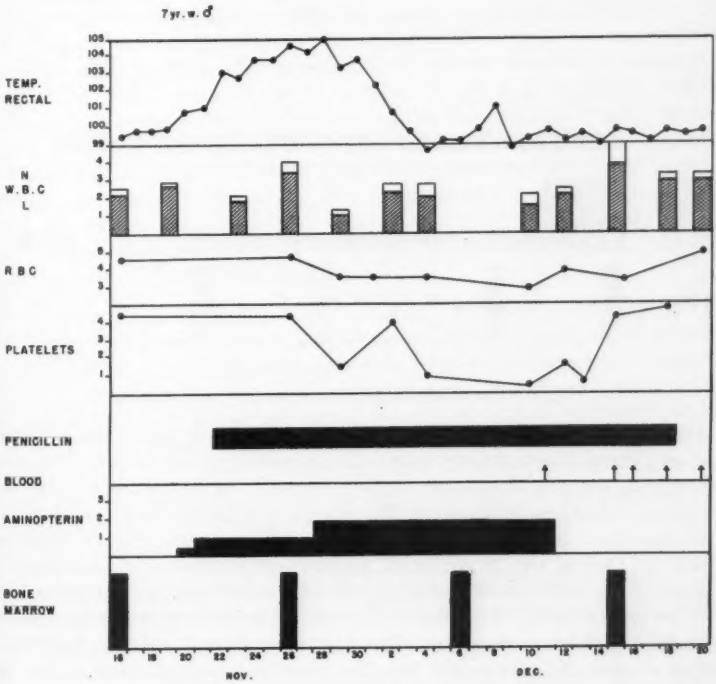


Figure 2 (case 6). Clinical response only.

During the first 21 days of her hospitalization she received 33 mg. of aminopterin, after which she complained of abdominal pain, pain on swallowing, and cracked, bleeding lips. The latter were interpreted as toxic manifestations and the drug was discontinued. She was also given penicillin daily and sufficient whole blood to keep the hemoglobin at a normal level. One week after starting therapy there was no change clinically, in the peripheral blood, or in the bone marrow. At the end of 2

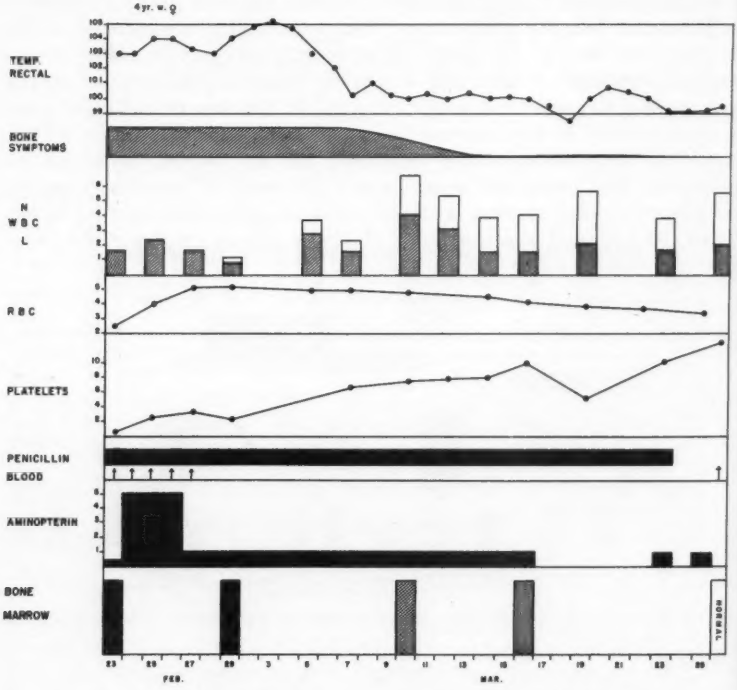


Figure 3 (case 12). Complete temporary remission.

weeks her temperature was subsiding, she was much more approachable, the peripheral blood showed increasing neutrophils and more mature lymphocytes, and the bone marrow also showed the appearance of granulocytes and fewer leukemic cells. At the end of the third week these findings had progressed, but she showed evidence of aminopterin toxicity. During the fourth week without aminopterin she continued to improve, ate well, wanted to play with other children, was afebrile, and the peripheral blood was practically normal and the bone marrow could not be considered abnormal. She was placed on 1 mg. aminopterin 3 times a week and discharged from the hospital to be followed as an outpatient where she has continued in good remission 58 weeks after starting therapy (fig. 3). This patient is representative of those few in whom there is complete temporary remission.

DISCUSSION

Although this series is too small to permit definite conclusions concerning the responsiveness to one or the other drug, the findings closely parallel those of other series. There seems little doubt that the total duration of the disease is prolonged by the present methods of treatment. In a series of 50 patients *(12)* treated prior to the use of these drugs only 12 percent survived for more than 6 months. In our series 53 percent survived for more than 6 months. In 300 children observed prior to the use of these drugs, spontaneous temporary remission was reported in 10 percent *(4)*. Temporary remission of symptoms and bone marrow findings in our series has been 6 times this expected spontaneous remission rate.

The exact mechanism by which the hormones and the folic acid antagonists produce remission is not known, but their action would appear to be different because the response is not parallel. The one may produce an initial response when the other does not, or may produce a response after resistance has developed to the other. It appears from this small series that the giving of hormones first, followed by aminopterin, may be the most effective regimen to follow. A combination of the two given concurrently does not appear to be more effective in patients with acute fulminating leukemia who fail to respond to treatment and die after a short course. It is not possible, however, consistently to predict which patients will respond to any form of treatment. Hoping that an earlier response might occur in the fulminating form of the disease through the use of an accelerated dosage of aminopterin, an amount of aminopterin estimated to produce a response has been tried in 3 patients, with good response in only 1 and possible earlier death in 2. Because of the variable amount of drug needed to produce response and the danger of a lethal dosage of a toxic drug, this method of accelerated treatment is not recommended.

Because of the frequency of serious infections occurring in the leukemic child, antibiotics should be part of the regimen in the acute febrile stage and during periods of drug toxicity. Septicemia, deep subfascial abscess, and severe pharyngitis have been complications in 3 patients while in temporary remission, all of whom responded satisfactorily to indicated antibiotic therapy. It is believed that serious infection would be of more common occurrence during periods of depressed bone marrow activity if antibiotics were not used routinely. Slight, but significant increase in survival time is reported in other studies by improved supportive measures in recent years *(13)*.

Blood is a necessary adjunct in the treatment. Large amounts may be required to tide the patient over if aminopterin intoxication is severe

(12) Cooke, J. V.: In Brenneman's Practice of Pediatrics. W. F. Prior Co., Hagerstown, Md. 1948. vol. III, chap. 18, p. 9.

(13) Southam, C. M.; Craver, L. F.; Dargeon, H. W.; and Burchenal, J. H.: Study of natural history of acute leukemia with special reference to duration of disease and occurrence of remissions. Cancer 4: 39-59, Jan. 1951.

and resulting bleeding is increased. It is not believed that exchange transfusions have any advantage over adequate amounts of blood given by the usual methods.

Irradiation locally to an infiltrative lesion resulting in pain, appears to give quick relief and in such patients is recommended. A vertebral lesion which had caused severe radiculitis was so treated with gratifying results.

SUMMARY

A series of 15 patients with acute leukemia was treated with some combination of aminopterin, ACTH, cortisone, blood, antibiotics, and roentgen rays. The response was defined as no response, clinical response only, and clinical response with complete temporary hematologic and bone marrow remission. It is suggested from this small series that the alternate use of hormone and aminopterin will produce the most satisfactory results. Antibiotics and blood transfusions are important supportive measures. The increased longevity as reported by others was closely paralleled by this series. Although no cure exists today for the leukemic child, dangerous thinking may result from considering the tools we have as only experimental. We should continue their use until more adequate therapeutic measures are found.

BOOK REVIEW

A Course in Practical Therapeutics, by *Martin Emil Rehfuss*, M. D., F. A. C. P., Professor of Clinical Medicine and Sutherland M. Prevost Lecturer in Therapeutics, The Jefferson Medical College, Philadelphia; Attending Physician, The Jefferson Medical College Hospital, Philadelphia, and *Alison Howe Price*, A. B., M. D., Associate Professor of Medicine, The Jefferson Medical College, Philadelphia; Asst. Physician to the Jefferson Medical College Hospital, Philadelphia; Chief of Diabetic Clinic, Curtis Clinic, Philadelphia. 2d edition. 938 pages; illustrated. The Williams & Wilkins Co., Baltimore, Md., publisher, 1951. Price $15.

This compendium of the course in clinical therapeutics at Jefferson Medical College has been revised and brought up to date. Since the appearance of the first edition of this reference book, F. Kenneth Albrecht has died. His article on fever is contained in the new edition. The schematic method of portraying each step in the treatment of disease has been retained. There are 4 main sections: (1) general therapeutic principles; (2) symptomatic therapy; (3) treatment of specific disorders; and (4) special treatment, including the therapeutic considerations of atomic warfare. The book is illustrated by schematic drawings and colored plates. The index is excellent, and there are useful tables for reference throughout the volume. This volume, like its predecessor, will serve as a valuable ready reference or guide for the general practitioner and specialist alike.

—*Commander T. D. Cuttle, MC, U. S. N.*

Lymphoblastoma in an Infant

Milton Kurzrok, *Commander, MC, U. S. N.* (1)

Francies Marshall, *Lieutenant, MC, U. S. N.* (1)

ACCORDING to Ewing (2), "not a few lymphosarcomas arise in infants." We believe, however, that the patient here reported is of interest because the initial roentgenographic findings were of such a nature as to be easily confused with those of benign thymic hypertrophy. Furthermore, earlier recognition of this condition is desirable from the standpoint of treatment and prognosis even though the ultimate outcome remains unaltered.

CASE REPORT

A mongoloid white girl, 2 4/12 years old, was admitted to this hospital on 1 October 1950 because of respiratory distress of 3 days' duration. Her mother stated that she had had a head cold 2 weeks previously and that she had only partially recovered from this infection. The family physician examined the patient shortly after the onset of dyspnea and gave her an injection of penicillin and epinephrine; in addition, he prescribed a sulfonamide to be taken by mouth. Temporary improvement was noted, but the dyspnea recurred and became progressively more severe.

The admission physical examination revealed an acutely ill child who presented the classical features of mongolism. Her rectal temperature was 103° F. She was in marked respiratory distress with stridor and infrasternal retraction. Slight cyanosis was present. There was evidence of an acute infection of the upper respiratory tract with involvement of the middle ears and the nasal and pharyngeal mucosa, but the most striking finding was the presence of huge, inflamed palatine tonsils which met in the midline, almost obstructing the airway. Numerous, firm, nontender anterior cervical lymph nodes were present. On percussion of the chest, there was definite sternal and parasternal dullness. Auscultation revealed a diminution in breath sounds throughout, more marked anteriorly; a few rhonchi were heard but no true rales.

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(1) U. S. Naval Hospital, Oakland, Calif., at time of writing this article; now stationed at U. S. Naval Hospital, Chelsea, Mass.

(2) Ewing, J.: Neoplastic Disease. 4th edition. W. B. Saunders Co., Philadelphia, Pa., 1940. p. 431.

The white blood cell count was 25,400, with a moderate shift to the left. There was a mild hypochromic anemia. Throat culture was reported to reveal a profuse growth of Monilia spores. The admission roentgenograms of the chest (fig. 1) showed marked superior mediastinal enlargement chiefly on the left side but to some extent on the right; in addition, an irregular infiltration was noted in the right upper lobe of the lung. A swallow of barium sulfate accomplished 10 days later showed no evidence of tracheal or esophageal compression or deviation.

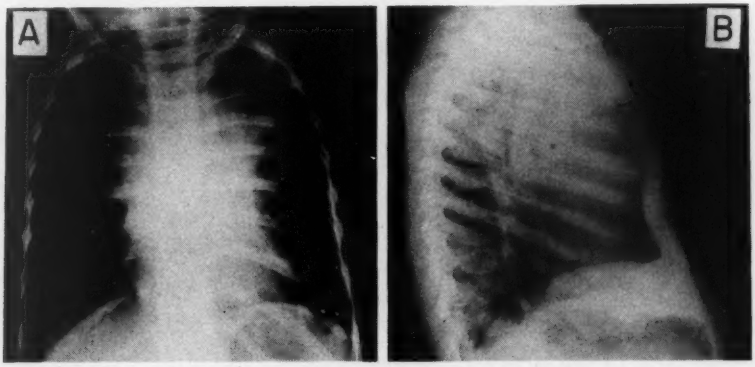

Figure 1. (A) *Posteroanterior view of the chest showing the marked superior mediastinal enlargement, chiefly on the left side. There is also evidence of an infiltrative process in the right upper lobe of the lung.* (B) *Right lateral view of the chest showing the lack of aeration in the anterior superior pulmonary region as a result of replacement by the main tumor mass.*

The child was placed in an oxygen tent on admission and the cyanosis disappeared promptly. In an effort to combat the pharyngeal and pulmonary infection the patient was given 50,000 units of penicillin every 3 hours and 250 mg. of terramycin every 6 hours. Tracheotomy was considered but was deferred until the response to other measures could be evaluated. By the fourth hospital day her temperature had returned to normal and the dyspnea had subsided; oxygen was no longer required. Because it was the consensus that the mediastinal mass represented an enlarged thymus, x-ray therapy was started on the second hospital day, and 2 treatments of 100 r. each were given over the anterior mediastinum. The child's general condition remained improved during the following week, but because the nature of the mediastinal lesion was undetermined two small anterior cervical lymph nodes were excised for microscopic study on the eleventh hospital day. The sections revealed lymphoblastoma.

Several days after the biopsy had been performed the patient's fever, dyspnea, and signs of intoxication returned. She died on the seventeenth hospital day while a tracheotomy was being performed to relieve

the extreme dyspnea. The immediate cause of death appeared to be respiratory obstruction.

Autopsy findings. There was a large, white, firm mass in the anterior mediastinum, measuring 9 by 10 by 4 cm. in its bulkiest proportions, and extending up into the neck bilaterally. No thymus gland per se could be found. The periaortic, mediastinal, peritracheal, and cervical lymph nodes were all enlarged and had the same firm consistency and white color as the intrathoracic tumor. Grossly, the trachea was not invaded by the malignant process nor did it appear compressed at any point. There was bilateral pleural effusion.

On microscopic examination the mediastinal mass presented a diffuse, homogenous collection of closely packed large lymphocytes. Scattered areas of caseation necrosis were present throughout the tumor mass. There was no evidence of thymic tissue or architecture to suggest primary involvement of this organ. Similar lymphocytic invasion was also seen in the lymph tissue generally, with greatest involvement in the tonsils, spleen, appendix, and the periaortic and mediastinal lymph nodes. Less extensive infiltration was also noted in the visceral pleura, lungs, and liver. A histologic diagnosis of malignant lymphoma, lymphosarcomatous type was made.

DISCUSSION

Massive mediastinal enlargement in children should always suggest the possibility of lymphosarcoma. Indeed, Wiglesworth et al. *(3)* in their report of 3 such patients, stated that this malignancy is actually the most common cause of such enlargement. Their youngest patient was 6 years of age, while our patient was just 2 4/12 years old. Furthermore, they were able to identify thymic tissue and the architecture in 2 of their patients and concluded that the thymus was the primary site of involvement. We were unable to reach such a conclusion regarding our patient because the main mediastinal mass on section bore no resemblance to the thymus. It would seem logical, however, to believe that this gland was completely replaced by the tumor tissue and it may well have been the initial site of the lesion.

Many other recent reports emphasize the fact that this disease is far from rare in the young. Craver *(4)*, in a survey of the general group of so-called lymphomas in children admitted to the New York Memorial Hospital since 1931, reported 7 patients with lymphosarcoma. The age of onset varied from 3.3 to 15 years in his series. Two percent of the 477 patients with lymphoblastoma reported by Minot and Isaacs *(5)* were shown to have an onset at between birth and 4 years of age.

(3) Wiglesworth, F. W.; Childe, A. E.; and Goldbloom, A.: Mediastinal lymphosarcomas in childhood. J. Pediat. 9: 331-349, Sept. 1936.

(4) Craver, L. F.: Lymphomas, leucemias, and allied disorders in children. J. Pediat. 15: 332-339, Sept. 1939.

(5) Minot, G. R., and Isaacs, R.: Lymphoblastoma (malignant lymphoma); age and sex incidence, duration of disease, and the effect of roentgen-ray and radium irradiation and surgery. J. A. M. A. 86: 1185-1189, Apr. 17, 1926; cont. 86: 1265-1270, Apr. 24, 1926.

Most modern clinicians believe that simple thymic hypertrophy plays a minor or even negligible role in the genesis of respiratory embarrassment (6). Although the extreme dyspnea which was present in our patient should have aroused our suspicions as to the nature of the mass, the huge tonsils further confused the issue in that we at first believed that they were probably contributing to the obstruction. Thus lymph node biopsy and the true diagnosis were unnecessarily delayed. Fortunately, the disease was of such a nature and degree that the delay did not effect the final outcome.

SUMMARY

Lymphoblastoma should be suspected in children with massive mediastinal enlargements. A review of the recent medical literature indicates that malignant lymphoma in children is not rare.

(6) Holt, L. E., and Howland, J.: Holt's Diseases of Infancy and Childhood. 11th edition. D. Appleton-Century Co., Inc., New York, N. Y., 1940. pp. 680-681.

BOOK REVIEW

Antibiotic Therapy, by *Henry Welch*, Ph. D., Director, Division of Antibiotics, Food and Drug Administration, Federal Security Agency of the United States Government, and *Charles N. Lewis*, M. D., Medical Officer, Division of Antibiotics, Food and Drug Administration, Federal Security Agency of the United States Government. Foreword by *Chester S. Keefer*, M. D., Wade Professor of Medicine, Boston University School of Medicine; Chairman, Committee on Medicine and Chairman, Committee on Chemotherapy of the National Research Council. 562 pages. The Arundel Press, Inc., Washington, D. C., publisher, 1951. Price $10.

This book discusses briefly the discovery and development of the major antibiotics. It summarizes a multitude of current medical reports of the various diseases treated, doses used, and the results obtained with each antibiotic. An adequate bibliography follows each chapter. In the first half of the book the use of tyrothricin, penicillin, streptomycin, bacitracin, aureomycin, chloramphenicol, and terramycin is discussed. In the last half of the book the treatment of various conditions such as tuberculosis, urinary and gastrointestinal tract infection, virus and rickettsial diseases is discussed. A brief summary of essential clinical features of some of these diseases is included. To the specialist these may be superfluous but they will be of great value to the general practitioner. This book should be in all medical libraries and most general practitioners, internists, and pediatricians will want it in their personal libraries. It is unfortunate that this sort of compilation becomes obsolete so rapidly. It is hoped that the authors will issue an annual or biennial supplement to keep the book up to date.

—*Col. M. C. Davidson, MC, U. S. A.*

Traumatizing Foreign Body in the Esophagus

Robert T. Gants, *Colonel, MC, U. S. A. (1)*

RETAINED foreign bodies in the esophagus usually prove fatal if not removed. At all military installations there is the ever present possibility that a patient may require treatment for removal of a foreign body lodged in the esophagus. When necessary, these patients should be transported quickly and safely to a hospital where the services of an oral endoscopist are available. To wait for spontaneous expulsion is dangerous. Blind probing, even with fluoroscopic visualization, in an attempt to dislodge or push the object on into the stomach is extremely hazardous and should not be attempted. Most foreign bodies that become lodged in the esophagus should be removed by peroral esophagoscopy.

Jackson and Jackson *(2,3)* stated that foreign bodies can be removed by esophagoscopy through the mouth in almost all cases, it never being necessary in their experience of over 1,500 cases to remove a foreign body by external esophagotomy except in a single instance in which there was stenosis due to pulsive and tractive implantation. Temple *(4)* preferred a transpleural approach for removal of certain foreign bodies in the esophagus, repair of the esophagus, and drainage of the mediastinum rather than the posterior extrapleural approach. Sweet *(5)* stated that if esophagoscopic measures fail to remove the foreign body, a direct approach may be necessary through a standard thoracotomy incision.

With the development of thoracic surgery, endotracheal anesthesia, and the use of antibiotics, the esophagus is becoming more and more

(1) 98th General Hospital.

(2) Jackson, C., and Jackson, C. L. (editors): Diseases of the Nose, Throat and Ear, Including Bronchoscopy and Esophagoscopy. W. B. Saunders Co., Philadelphia, Pa., 1945. pp. 745-747.

(3) Jackson, C., and Jackson, C. L.: Bronchoesophagology. W. B. Saunders Co., Philadelphia, Pa., 1950. p. 26.

(4) Temple, L. J.: Treatment of perforation of esophagus; report of 3 cases. Brit. M. J. 1: 935-937, May 28, 1949.

(5) Sweet, R. H.: Thoracic Surgery. W. B. Saunders Co., Philadelphia, Pa., 1950. p. 235.

amenable to indicated surgical treatment. Transthoracic esophagotomy for the removal of an impacted and traumatizing foreign body is now sometimes safer than so-called conservative attempts at removal through esophagoscope with almost certain trauma or perforation and resultant mediastinitis. This is especially true when the services of a surgeon who is skilled in removal of foreign bodies through the esophagoscope are not available. The following case is reported as an example.

CASE REPORT

A 17-year-old soldier was admitted to this hospital on 13 February 1950, having been transferred by airplane. At about midnight on 11 February he had suddenly awakened and noted bleeding from his throat. His removable denture, consisting of the upper 2 central incisors, from which a clasp and porcelain facing had been broken months before, which fitted none too snugly, and which he habitually wore while sleeping, had disappeared and seemed to be lodged deep in his throat. He immediately sought medical advice. Radiologic examination revealed the partial denture to be in the upper esophagus. At the local medical installation, no attempt at removal or instrumentation was made. He was not given any food or liquids by mouth. Penicillin was administered intramuscularly. Frequent radiologic examinations revealed that the foreign body had progressed downward to a point where it became immobile at the level of the seventh and eighth thoracic vertebras (fig. 1). The patient was in no distress.

The denture was about 5 cm. long and 1.5 cm. wide. There were 7 sharp, curved clasps which pointed in all directions so that they could easily imbed themselves in the esophageal wall, no matter in which direction the denture was moved (fig. 2). Through the esophagoscope, the denture was easily seen at the level noted on the roentgenograms. It could not be moved by gentle manipulation. Another esophagoscopic examination and unsuccessful attempt to remove or dislodge the foreign body was performed on 15 February, 35 hours after admission. The position of the denture had not changed. During this time, the patient's temperature and leukocyte count remained normal. He had expressed no complaints except for the discomfort of the esophagoscopic procedures.

A Levine tube was inserted through the nose, into the esophagus, and on into the stomach. It passed the lodged foreign body with ease. It was determined that the denture with its 7 curved and sharp clasps was firmly imbedded in the esophageal wall and that further endoscopic manipulative attempts would be unsuccessful and result in added trauma with possible perforation of the esophagus. Furthermore, to delay in removing the traumatizing foreign body would also result in eventual perforation and mediastinitis. It was decided to remove the foreign body by esophagotomy through the pleura.

At 1900 hours on 15 February, 45 hours after admission and 91 hours after swallowing the denture, the foreign body was removed by per-

Figure 1. Oblique projection of the chest revealing the position of the foreign body in the esophagus.
Figure 2. Foreign body removed from the esophagus.

forming an esophagotomy, employing a left transpleural approach. Positive pressure nitrous oxide and ether anesthesia were used. The operation required 3 hours. The left sixth rib was removed. The pleura appeared normal and was incised. After collapse of the normal left lung, the foreign body could be felt in a fixed position in the esophagus at the level of the seventh thoracic vertebra. There was no evidence of mediastinitis or perforation of the esophagus. The inferior pulmonary ligament and mediastinal pleura were divided and incised so as to expose the esophagus which was partially mobilized. Through a 3 cm. longitudinal incision in the esophagus, the denture was grasped. It was extracted with difficulty because the sharp clasps were deeply imbedded in the wall of the esophagus. The esophageal incision was closed in layers with interrupted 00 chromic catgut sutures on an atraumatic needle. An external buttress was fashioned from mediastinal areolar tissue and pleura. A posterior mediastinotomy was performed by making a stab wound in the eighth interspace posteriorly near the left vertebral border. A rubber tissue drain was inserted through this wound. The mediastinal pleura was then approximated. Two thoracotomy tubes were inserted through intercostal stab wounds. The chest was closed in layers using interrupted fine silk sutures. The thoracotomy tubes were connected to underwater seal bottles and the lung re-expanded. Penicillin and streptomycin were administered intrapleurally and intramuscularly. During the operation and immediately thereafter, the patient received 1,000 cc. of citrated whole blood.

The postoperative course was uneventful except for a chill and spiking fever on the second, fourth, and sixth postoperative days. It was noted that the febrile reaction occurred each time intravenous protein hydrolysate was administered. Roentgenograms of the chest on the first postoperative day revealed the lung to be expanded. The thoracotomy tubes were removed on the fourth postoperative day. The posterior mediastinotomy drain was completely withdrawn by the tenth postoperative day and the patient was eating a soft diet, the Levine tube having been removed 2 days before. He was returned to full military duty on 11 April after having spent the prescribed length of time at the Rehabilitation and Convalescent Center, during which time he was fitted with a permanent, fixed, dental prosthesis. A follow-up report 1 year later revealed that the patient is perfectly well. An esophagram made at his local station hospital was reported as normal.

SUMMARY

The successful management of a patient with a traumatizing foreign body imbedded in the esophagus which was removed by transthoracic esophagotomy is reported. Since this patient was operated on, another article *(6)* concerning esophageal operations, particularly about per-

(6) Samson, P. C.: Postemetic rupture of esophagus. Surg., Gynec. & Obst. 93: 221-229, Aug. 1951.

forations and management by thoracotomy, has been published. After perusal of this article and review of the case here reported, it seems that the performance of a mediastinotomy with drainage and the delay in removing the nasogastric suction tube was probably unwarranted.

BOOK REVIEWS

Surgery of Peripheral Nerves, by *Emil Seletz,* M. D., F. A. C. S., F. I. C. S., Assistant Clinical Professor of Neurological Surgery, University of Southern California, School of Medicine; Associate Senior Attending Neurosurgeon, Cedars of Lebanon Hospital; Junior Attending Neurosurgeon, Los Angeles General Hospital, Los Angeles, Calif. Art Editor, *Tom Jones,* Professor of Medical and Dental Illustration and Head of the Department, University of Illinois, Chicago, Ill. 185 pages; illustrated. Charles C Thomas, Publisher, Springfield, Ill., 1951. Price $10.75.

This is a new book based on the experience with 2,037 peripheral nerve injuries incurred in World War II. Injuries of the important peripheral nerves as well as cranial nerves VII, XI, and XII are discussed giving the anatomy, the dysfunction resulting from injury at the various levels, and the best surgical approach for repair. The book is exceptionally well illustrated including photographs of typical deformities and surgical exposures as well as anatomic drawings and diagrams. The latter are the work of Tom Jones, Professor of Medical and Dental Illustration at the University of Illinois, and maintain his usual high standard. The surgical approaches recommended are commendable and are designed to prevent disability from scar contracture and surgical trauma. An original rerouting of the posterior tibial nerve to gain additional length is presented, but too briefly to be of much value. The book is too elementary for established neurosurgeons, but is strongly recommended for general surgeons who occasionally perform peripheral nerve repairs. The well-illustrated surgical approaches especially deserve wide publicity.—*Commander D. W. Miller, MC, U. S. N.*

Textbook of Biochemistry, by *Edward Staunton West,* Ph. D., Professor of Biochemistry, University of Oregon Medical School, and *Wilbert R. Todd,* Ph. D., Associate Professor of Biochemistry, University of Oregon Medical School. 1,345 pages; illustrated. The Macmillan Co., New York, N. Y., publishers, 1951. Price $12.

The writing of a comprehensive textbook of biochemistry is difficult when the authors must anticipate readers inadequately grounded in the fundamentals of organic, physical, and colloid chemistry. Unfortunately many students would like to become biochemists without first becoming chemists and the authors of this text have made an excellent attempt to resolve this difficulty in their introductory chapters. The uninitiated should not, however, assume that this synopsis

is a substitute for a basic course in physical chemistry. Subsequent chapters on lipoids, carbohydrates, and proteins are developed in the traditional manner with up-to-date material. Half of the book deals with the chemistry of digestion, metabolism, respiration, body fluids, vitamins, and nutrition, all of which have been well presented.

As a textbook for the usual year's course in biochemistry, it is very long and tempts the instructor to cover too much ground too hurriedly. It is not an exhaustive reference book for the specialist, but it is recommended to biochemists and physicians with special interests in chemistry as an up-to-date, readable, and useful volume.

—*Lt. Col. M. E. Freeman, MSC, U. S. A.*

Surgery of the Oblique Muscles of the Eye, by *Walter H. Fink,* M. D., Minneapolis, Minn. 350 pages; 93 illustrations, including 18 in color. The C. V. Mosby Co., St. Louis, Mo., publisher, 1951. Price $8.75.

This is an excellent book for the ophthalmologist interested in ocular motility and operations on the eye muscles. The format is attractive and the book is profusely illustrated. The chapters are arranged in logical order and the content of each chapter is listed beneath its heading. The material is easy to locate in spite of the rather meager index. The book was designed to cover the oblique muscles and the associated factors necessary to understand the obliques and their functions. Part I covers embryology; developmental anomalies; and comparative microscopic, gross, and surgical anatomy. Part II includes a review of operations on the oblique muscles, the frequency and cause of muscular defects, the function of the obliques, the diagnosis of oblique muscular defects, indications for operation, and surgical technic. The greatest value of the book lies in its documentation of the great amount of work the author has done on the anatomy of the obliques and its application. He has given the average locations and the extreme variations in origins and insertions. He has carefully documented his material and has presented the viewpoints of numerous authorities in this field. In each instance, however, he has given his own opinion, based on his own experience. This book should be available to ophthalmic surgeons who work in the field of ocular motility.—*Col. V. A. Byrnes, U. S. A. F. (MC)*

Mediastinal Emphysema

Warren E. Klein, *Captain, MC, U. S. N.* (1)
William H. Neil, *Lieutenant, MCR, U. S. N. R.* (2)
John B. Stotler, *Lieutenant, junior grade, MC, U. S. N.* (3)
Ralph L. Eslick, *Lieutenant Commander, MC, U. S. N.* (2)

A RAPIDLY growing group of enthusiasts are participating in an aquatic sport which is not entirely without hazard. These hardy devotees of skin diving, or goggle fishing, on occasion may descend to depths of from 30 to 35 feet, investigating caverns and crevices beneath coral formations and stalking choice targets. In the excitement of the chase, and if the prey be wary, the skin diver may remain at depth until progressive anoxia forces a rapid return to the surface. This "crash surfacing" procedure requires close adherence to an acquired training pattern, exhaling as the ascent is made to prevent overexpansion of the lungs, which might otherwise produce a "Valsalva effect" (4) with syncope, or may cause rupture of an alveolar bleb and consequent mediastinal emphysema, air embolism, or spontaneous pneumothorax, should the pleura be involved. Two cases are reported here: one demonstrating trauma as described above, the second demonstrating trauma caused by ill-advised straining while in an abnormal position.

CASE REPORTS

Case 1. A 29-year-old man was admitted with a tentative diagnosis of retro-esophageal abscess. He had spent most of the day spear fishing and noticed that his throat felt somewhat tender on returning home. He had descended to depths of 30 feet repeatedly during the afternoon. While eating his evening meal he noted that the discomfort was aggravated by swallowing and described a "swelling" in the back of his

(1) U. S. Naval Air Station, Key West, Fla., when article was written. Currently staff medical officer, First Marine Aircraft Wing, Fleet Marine Force.
(2) U. S. Naval Air Station, Key West, Fla.
(3) U. S. Naval Hospital, Key West, Fla., when this article was written. Currently attending the U. S. Naval Medical School, National Naval Medical Center, Bethesda, Md.
(4) In the "Valsalva effect," high pressure is maintained within the respiratory system by fixed thoracic cage and diaphragm with a closed glottis while the subject rises from depth to the surface of the water and a lower ambient pressure. Vertigo and syncope may, and often do, follow. The experienced diver learns to open his glottis and exhale slowly as he ascends, thus maintaining equilibrium between contained and ambient pressures.

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throat. Later while reading, with his neck in a flexed position, he was again aware of the "swelling" and of discomfort in the back of his throat at about the level of the fifth cervical vertebra. He found that any movement of his neck intensified the discomfort in this region. No additional symptoms were noted other than a generalized sensation of warmth, apparently caused by mild sunburn. He had eaten no fish during the preceding month and he recalled no trauma to his throat. Physical findings included a mild first degree sunburn on the back, tenderness on displacement of the thyroid cartilage laterally or posteriorly, and induced pain at the level of the fifth cervical vertebra in the posterior midline on extreme anteflexion of the neck. The chest was clear, the nasal mucosa was mildly injected and the pharynx was normal. Pressure on the postpharyngeal wall produced no discomfort. The temperature was 99° F. The blood pressure was 112/80. The leukocyte count on admission was 16,500 with 70 percent segmented cells, 28 percent lymphocytes, 1 percent monocytes and 1 percent basophils. Crepitus was elicited over the anterior triangle of the neck, bilaterally.

A roentgenogram on admission showed an area of decreased density lying behind the esophageal shadow and extending from the oropharynx to well below the level of the thyroid cartilage, which was strongly suggestive of air dissecting tissue planes (fig. 1). Interstitial emphysema from infection or from a spontaneous mediastinal emphysema was postulated. The patient was transferred to the U. S. Naval Hospital for observation. Treatment was expectant. A roentgenogram of the cervical structures on the second day of illness revealed the previously described abnormally decreased density behind the esophageal shadow to be increased, with diffusion (fig. 2). An inspection of posterior, anterior and lateral films of the chest revealed no evidence of mediastinal emphysema or other abnormality. A roentgenogram on the fifth day revealed the prevertebral structures to be normal. The roentgenologist's impression was that the patient had a retro-esophageal emphysema which apparently had completely cleared.

After 3 days in the hospital the patient was asymptomatic and was discharged with the diagnosis: Emphysema, retro-esophageal, caused by trauma, incurred while skin diving to a depth of 30 to 35 feet, when an alveolar bleb apparently ruptured into the mediastinum.

Case 2. A 21-year-old man reported complaining of throat discomfort, dysphagia, and dysphonia. He had spent the morning catching spiny lobsters in shallow water, employing a diving face mask. Although he had at no time descended to a depth greater than 4 feet, he had leaned from a boat repeatedly with head and shoulders submerged in an inverted position for sufficient time to develop marked cyanosis. The onset of symptoms was gradual and first noted while propelling the boat toward shore by means of a pole.

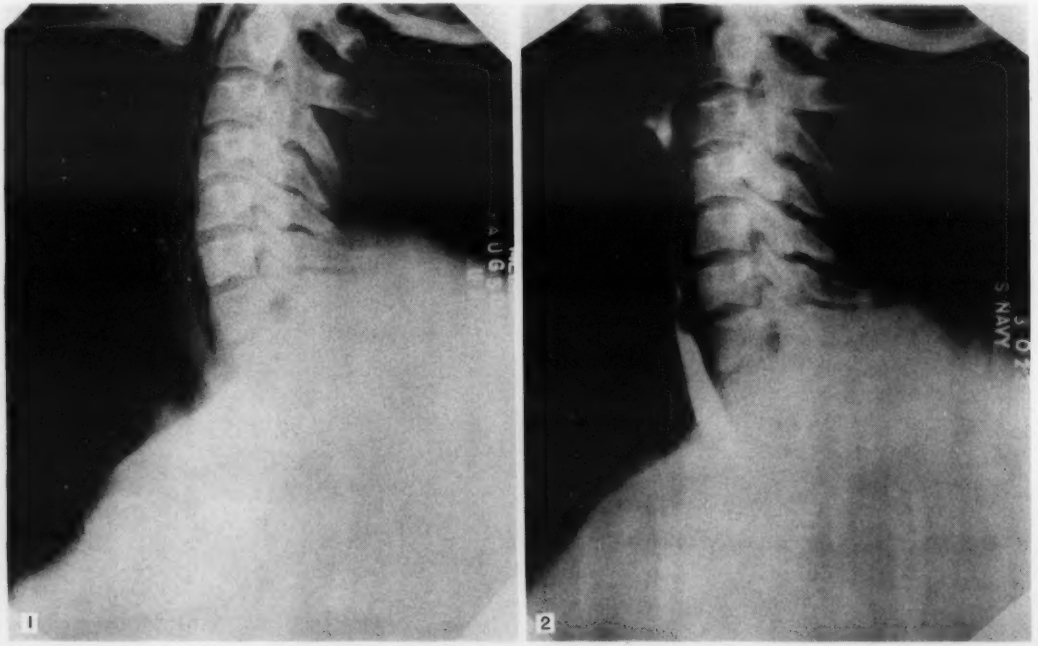

Figure 1 (case 1). Lateral cervical roentgenogram taken on admission showing interstitial emphysema of the postesophageal space.
Figure 2 (case 1). Roentgenogram, second day, showing barium in esophagus and an increase with diffusion in the amount of air in the postesophageal tissues.

Soreness of the throat was followed rapidly by pain in the chest, described as follows: "It felt like my lungs were inflamed." Deep breathing accentuated this pain. The patient felt that stretching his neck should reduce the pain but found that this maneuver produced no relief.

His temperature was 99.6° F; his pulse was 106; his respiratory rate was 20; and his blood pressure was 120/74. Pain was elicited by light percussion under the fourth left interspace at the costochondral junction. On auscultation of the heart, a slapping sound was heard in the tricuspid area. The patient was in acute distress and reported pain on opening his mouth and on depression of his tongue. His throat appeared normal. Soft tissue crepitus and tenderness were elicited bilaterally, on examination of his neck. There was mild swelling in both anterior triangles. Palpation of the right upper and lower abdominal quadrants caused sharp pains in the chest and neck.

The leukocyte count was 18,000 with 89 percent segmented cells, 1 percent stab cells, 6 percent lymphocytes, 3 percent monocytes, and 1 percent eosinophils. Radiographic studies revealed interstitial emphysema of moderate degree, involving the fascial planes of the neck and, less markedly, the mediastinal structures. Dissection by free air was noted beneath the parietal pleura overlying the right hemidiaphragm at the cardiophrenic angle and along a major bronchus in the left lung (figs. 3-6).

The patient was admitted to the sick list and observed. Fowler's position was found to give the best relief from respiratory embarrassment and pain. The following day crepitus was still apparent. The heart sounds were normal and abdominal tenderness was elicited in the left lower quadrant with deep tenderness on rectal exploration. The referred pain in the chest noted on admission was absent. Mild respiratory embarrassment persisted. The leukocyte count was 7,600. On the third day all symptoms had subsided. On the fourth day no radiographic evidence of emphysema was discernible. A diagnosis of emphysema, traumatic, caused by underwater fishing was made.

DISCUSSION

Mediastinal emphysema supervenes when alveolar blebs rupture into the mediastinal tissues and may give rise to quite alarming symptoms. Diving, with or without full equipment, with rapid ascent and neglect of safety precautions, may produce this syndrome as the ambient pressure decreases and the alveolar pressure is maintained. The experienced diver normally allows air to escape from the lungs without giving conscious thought to the process, but with the development of anoxia in a prolonged dive, without air or oxygen supply, the survival instinct dictates escape from the situation leading to the anoxic state, and a mild degree of hysteria is experienced. This may interfere with effective adherence to a training pattern. The subject closes his glottis

Figure 3. (case 2). Admission roentgenogram of chest demonstrating extra-alveolar air in the left lung along the left cardiac border and in the upper mediastinum. Note the air in the right leaf of the diaphragm. Figure 4 (case 2). Lateral cervical roentgenogram taken on admission showing interstitial emphysema of the postesophageal space.

Figure 5 (case 2). Roentgenogram on day of admission showing air in the upper mediastinum. Figure 6 (case 2). Lateral roentgenogram of the chest made on admission demonstrating air dissecting the peribronchial tissues of the lung and free air beneath the parietal pleura of the diaphragm.

and holds his breath, despite his understanding of the mechanism of safe approach to lower pressure levels. The excitement of the chase may contribute to this tendency to hold the breath. The relative intrapulmonary pressure rises and a weak bleb wall ruptures.

The second case was precipitated by exercise and strain while in an inverted position with head and shoulders submerged. Although this was also a Valsalva effect, no great ambient pressure change was experienced. Overenthusiasm was responsible in great degree.

The more common injuries incident to skin diving include sunburn of various degrees, minor lacerations and punctured wounds, coral abrasions, various stings and locally toxic injuries produced by coelenterates and plants encountered on the reefs, none of which deter the participants nor, indeed, is this newly recognized hazard likely to do so.

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Pronestyl in the Treatment of Ventricular Tachycardia[1]

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William H. Isham, *Captain, MC, U. S. A.*

PROCAINE amide hydrochloride (pronestyl) has recently been advocated for use in the treatment of ventricular tachycardia (2). The drug can be administered orally or intravenously. A report of a case of paroxysmal ventricular tachycardia treated with both oral and intravenous pronestyl follows.

CASE REPORT

A 24-year-old man was admitted to this hospital on 24 May 1951 complaining of tachycardia occurring over a period of 18 months prior to admission. There was no history of cardiac disease or rheumatic fever. These attacks of tachycardia were accompanied by dyspnea, pounding noises in the ears, vertigo, and a sensation of impending syncope. The symptoms would last for a few minutes and then disappear but the tachycardia would persist for periods varying from 1 hour to 1 week. Usually the attacks would last from 1 to 2 hours. Pressure on the eyes, ice packs to the chest, mild sedatives, and digitalis were all tried without lasting results. On the day prior to admission, the patient had an attack of tachycardia which lasted about 24 hours. He stated that he smoked 1 or 2 packages of cigarettes a day and that his alcoholic intake was small.

His blood pressure was 124/88. His heart was not enlarged to percussion and no murmurs or thrills were noted. On admission the rhythm was normal and the rate was 85. An ECG on the day of admission revealed ventricular extrasystoles, a sinus tachycardia, and inverted T-waves in leads 2, 3, AVF, V_5, and V_6. On the following day an ECG showed ventricular tachycardia. ECG's immediately following the cessation of the tachycardia revealed inversion of the T-waves in all limb

(1) Tripler Army Hospital, Honolulu, T. H.

(2) Mark, L. C.; Berlin, L.; Paden, H. J.; Rovenstein, E. A.; Steele, J. M.; and Brodie, B. B.: Action of procaine amide (N_1-(2-di-ethylamino-ethyl)-P-amino-benzamide) in ventricular arrhythmias. J. Pharmacol. & Exper. Therap. 98: Jan. 21, 1950.

leads and in the precordial leads V_4, V_5, and V_6 (fig. 1). The patient had 2 subsequent attacks of ventricular tachycardia in the hospital. By 12 July the T-waves in the limb leads were assuming a more normal pattern. The T-waves in leads V_4 and V_5 were still slightly inverted (fig. 2).

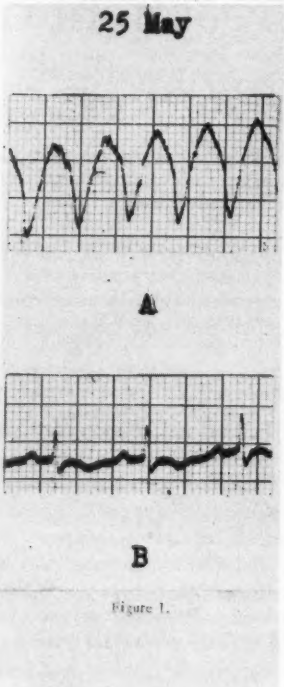

Figure 1.

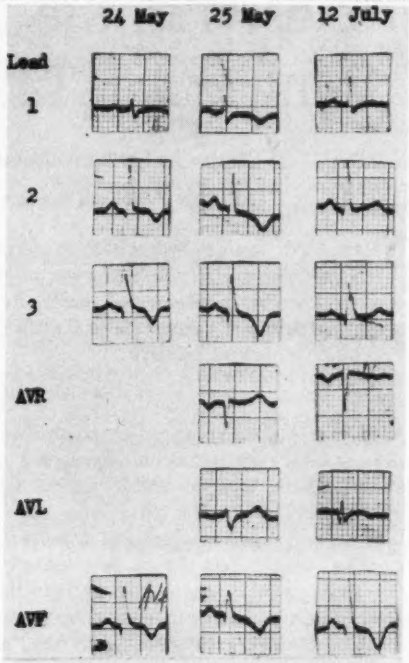

Figure 2.

Figure 1. (A) *Lead 1 taken during ventricular tachycardia.* (B) *Lead 1 taken 1 minute after intravenous injection of 100 mg. of pronestyl. Figure 2. ECG's illustrating T-wave changes that occurred during hospitalization. 24 May tracings were taken at the time of admission. 25 May tracings were taken immediately following an attack of ventricular tachycardia and an intravenous injection of pronestyl. 12 July tracings were taken after 6 weeks' bed rest and gradual ambulation with the patient on a maintenance dose of pronestyl.*

On the day following admission immediately after completing an electrocardiographic tracing with a normal sinus rhythm, the patient began to smoke a cigarette. Within 1 minute of lighting the cigarette, he noted the symptoms associated with previous attacks of tachycardia. An ECG was made at that time and it was noted that the patient was having an attack of ventricular tachycardia. One hundred milligrams of pronestyl were administered intravenously. Within 3 minutes a normal sinus rhythm was restored. The patient was placed on a maintenance dose of

0.5 gram of pronestyl by mouth every 6 hours. Four hours later and after his first oral dose of pronestyl, he developed another attack of ventricular tachycardia. Again, after the intravenous administration of 100 mg. of pronestyl, the tachycardia stopped within 1 minute and a normal sinus rhythm was restored. ECG's thereafter showed T-wave inversion in all limb leads and in leads AVF, V_4, V_5, and V_6. The patient was continued on an oral dose of 0.5 gram of pronestyl every 6 hours. Four weeks later he had a recurrence of tachycardia, during gradual reduction of the oral dosage of pronestyl and the institution of quinidine therapy. The attack was immediately controlled by giving 100 mg. of pronestyl intravenously. With the resumption of a dosage of 0.5 gram of pronestyl by mouth every 6 hours, no further attacks occurred. Other treatment of this patient consisted of bed rest for 6 weeks and gradual ambulation. At the time of discharge, he was on a maintenance dose of 0.5 gram of pronestyl every 4 hours.

DISCUSSION

We believe that this patient's tachycardia was precipitated in part by the use of tobacco because he was a heavy smoker; his first attack of tachycardia in the hospital came on while he was smoking a cigarette. It is also of interest to note that he was having attacks of paroxysmal ventricular tachycardia from which he could spontaneously recover and carry on his duties for 18 months prior to admission. The T-wave changes in the ECG's showing sinus rhythm were thought to be due to myocardial fatigue although myocardial infarction was considered. The use of pronestyl appeared to control the ventricular tachycardia in this patient. When used intravenously, the drug acted rapidly to produce the desired effect. The dosage used was much smaller than usually reported. In a 2-month period of oral administration there were no apparent signs of toxicity. The experience noted herein is similar to that reported by other observers (3,4) in that quinidine failed to prevent the recurrence of attacks.

(3) Irvin, C. W., Jr., and Cutts, F. B.: Ventricular tachycardia; report of case in which "pronestyl" was effectively used after failure with quinidine. J. A. M. A. 146: 1282-1283, Aug. 4, 1951.

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Fluid Balance in Pediatrics[1]

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C. Warren Bierman, *First Lieutenant, MC, U. S. A. R.*

COINCIDENT with the rapid increase in the Armed Forces and the concomitant rise in dependent medical care, pediatric practice is becoming a feature of military medicine, many children being cared for at the dispensary and station hospital level by physicians without special pediatric training. The medical officer used to adults frequently finds himself at a loss on encountering fluid balance problems in children, in whom parenteral therapy is frequently required. The following concepts of fluid balance, which are taught as part of the intern teaching program of the pediatric section of this hospital, enable the house officer with limited pediatric experience to administer fluids with safety. Although dogmatic routines cannot provide optimum therapy in every situation, the procedures outlined will provide safe limits for those whose lack of experience does not provide facility to individualize.

GENERAL PARENTERAL REQUIREMENTS

In considering the fluid needs of a child on complete parenteral therapy, it is useful to consider the relationships of total fluid volume, extracellular fluid volume, and blood volume in proportion to body weight. Recent studies indicate that from 70 to 83 percent of the bodies of premature and newborn infants are composed of water, with a gradual decrease to between 53 and 63 percent in the ages from 6 months to 11 years *(2)*. Forty percent of the body weight of premature infants, 35 percent of that of full-term infants, and 30 percent of that of children from 1 to 14 years of age consist of water in the extracellular compartment *(3)*. The blood volume of infants up to 1 year of age is about 8 percent of the body weight, increasing to about 8.5 percent in the ages from 1 to 13 years *(4)*.

(1) Madigan Army Hospital, Tacoma, Wash.

(2) Friis-Hansen, B. J.; Holiday, M.; Stapleton, T.; and Wallace, W. M.: Total body water in children. Pediatrics 7: 321-327, Mar. 1951.

(3) Perley, A.; Forbes, G. B.; and Pennoyer, M. M.: Determination of sodium ^{24} "space" in infants, children, and adults. J. Pediat. 38: 299-305, Mar. 1951.

(4) Russell, S. J. M.: Blood volume studies in healthy children. Arch. Dis. Childhood 24: 88-98, June 1949.

Basic requirements. An infant needs an average of 120 cc. of water per kilogram of body weight, the need being greater in smaller infants, less in larger. He needs about 0.3 gram of sodium chloride per kilogram, an average of 1 gram per day for the infant and 3 grams per day for the child *(5)*. Additional amounts of water and sodium chloride must replace fluids lost through the removal of intestinal secretions by gastric suction, vomiting, or diarrhea. Four grams of dextrose per kilogram of body weight is sufficient to keep the patient free of ketosis and provide for some of his basic energy requirements. If nourished for more than 2 or 3 days parenterally, from 1.5 to 3 grams of protein per kilogram of body weight should be supplied either as blood, if the hemoglobin is

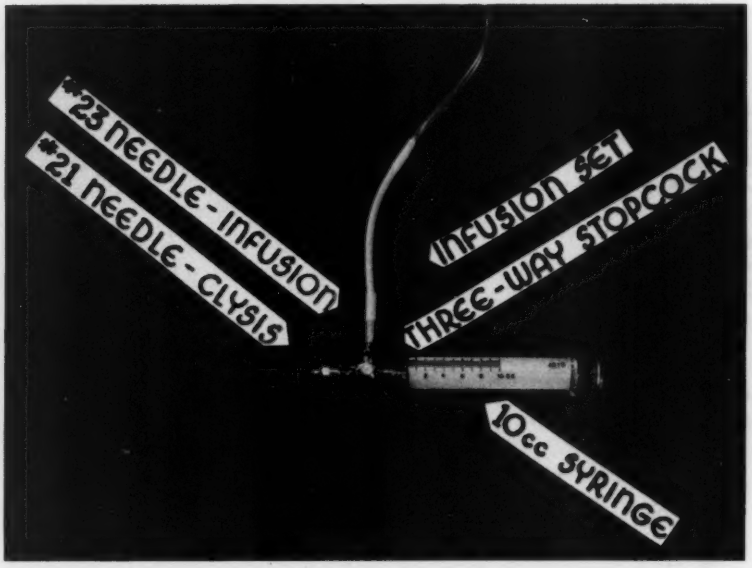

Figure 1. Apparatus for intermittent fluid administration under pressure.

low, or as salt-poor albumin, pooled plasma being avoided because of the danger of homologous serum jaundice and protein hydrolysates because they are not utilized as well as blood products. It is further desirable to provide 100 mg. of vitamin C per day either intravenously or orally and 25 mg. of vitamin K per day intramuscularly, especially if the patient has had a recent operation. One ampule of B complex vitamins may be mixed with the day's parenteral fluid requirements.

Method of administration. As a standard solution, a mixture of 2 parts 5 percent dextrose and 1 part normal saline solution, when given

(5) Butler, A. M., and Talbot, N. B.: Medical progress; parenteral-fluid therapy; estimation and provision of daily maintenance requirements. New England J. Med. 231: 585-590, Oct. 26, 1944.

in quantities sufficient to maintain fluid balance, provides adequate sodium chloride and dextrose and may be used either intravenously or subcutaneously. It is most important on a ward with changing nurses to mix only a 24-hour requirement at one time, especially if administration is continuous. This is a safeguard against giving too much fluid. For intermittent fluid administration, hypodermoclyses up to 40 cc. per kilogram may be given subcutaneously under pressure; infusions up to 30 cc. per kilogram may be given intravenously under pressure; transfusions up to 10 cc. per kilogram may be given under pressure with safety. Figure 1 shows a convenient and satisfactory apparatus for intermittent fluid administration. After administering a hypodermoclysis under pressure, the patient should not be left until the pink color has returned to the skin over the hypodermoclysis.

Drip hypodermoclyses and infusions have an advantage over administration under pressure in that the fluid is administered uniformly over a spaced period of time. They are potentially dangerous, however, unless carefully supervised. The doctor responsible for the patient, unless familiar with the nurses' reliability, should himself check the rate of flow frequently. Furthermore, the rate of flow and level remaining in the bottle should be recorded by the nurses every 30 minutes. If these safeguards cannot be fulfilled, we recommend spaced hypodermoclyses and infusions under pressure. Although in our hands hyaluronidase has been of little value in infants, the injection of 1 ampule of this substance into the tubing near the hypodermoclysis needles has proved of value in increasing the rate of hypodermoclysis absorption in children. Drip clyses may be run at a rate of from 4 to 8 drops per minute and drip infusions at 10 drops per minute. Up to 20 cc. per kilogram of body weight of whole blood may be given in this way to an infant at one administration and may be administered through a 23- or 25-gage needle.

Although the regimen outlined falls short of supplying minimal theoretical caloric requirements, it is adequate for the usual infant in whom total parenteral therapy is needed only for short periods. On employing these technics recently in a newborn infant who was unable to take food or fluid by mouth because of a congenital intestinal abnormality, his general nutrition remained excellent in spite of 2 major operations. At the time of death on the twenty-eighth day of his life he was well nourished and had regained his birth weight.

CORRECTION OF INITIAL DEHYDRATION AND ACIDOSIS

The infant who is dehydrated and acidotic presents additional problems of initial water and electrolyte deficits plus acid-base disturbances. If laboratory facilities are available, blood should be taken immediately for chemical examination and for crossmatching. The determinations found most useful are (1) blood urea nitrogen or nonprotein nitrogen to ascertain renal status (if elevated, administer fluids cautiously); (2) carbon dioxide combining power; (3) serum chloride; (4) if facilities are available, serum sodium and potassium to determine the

status of electrolyte and acid-base balance; and (5) total protein to determine the patient's initial nutritional state.

As soon as blood is drawn the child should receive initial fluids. For initial repair a solution containing 3 parts of 5 percent dextrose, 2 parts of normal saline, and 1 part of M/6 sodium lactate (3:2:1) provides a relatively physiologic sodium to chloride ratio and an excess of water (6). In the dehydrated child, an initial infusion of 30 cc. per kilogram under pressure and a hypodermoclysis of from 20 to 40 cc. per kilogram usually provides sufficient fluid and electrolytes until initial chemical determinations are reported. When urinating, and out of shock, up to 30 cc. of whole blood per kilogram may be given to the very ill or anemic patient. After initial hydration, the 24-hour maintenance requirements mentioned above should be given and therapy should be modified on the basis of chemical determinations and the general clinical condition.

Although the usual types of dehydration resulting from diarrhea, pyloric stenosis, and intestinal obstruction respond to this form of therapy, several metabolic disturbances, such as diabetic acidosis and salicylism, produce rather severe refractory acidoses which require more vigorous fluid therapy. In both, larger amounts of fluid, up to 150 cc. per kilogram of 3:2:1 solution should be given within the first 12 hours. If the initial carbon dioxide combining power is below 25, additional alkali in the form of M/6 sodium lactate may be given, remembering that 60 cc. per kilogram of body weight will ordinarily raise the carbon dioxide combining power by about 33 volumes per 100 cc. (7). Usually it is wise to give only one-half the calculated quantity of alkali in one administration.

Severe salicylism is frequently refractory to treatment, abnormal fat metabolism and metabolic acidosis persisting as long as an appreciable salicylate blood level remains (8). After the period of initial parenteral hydration, recovery has been expedited by offering 3:2:1 solution by mouth ad lib, within the bounds of calculated fluid requirements, in addition to continued parenteral therapy.

ROLE OF POTASSIUM IN PARENTERAL FLUID ADMINISTRATION

The administration of potassium must be considered when the patient has been maintained parenterally for periods of longer than 3 days, or in the recovery phase of acidosis. The known physiologic effects of potassium (9) are related largely to extracellular concentration. Clinical symptoms of potassium deficiency, associated with hypokalemia, are

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(8) Feldman, H. T.: Methyl salicylate poisoning. Clin. Proc. Child. Hosp. 6: 362-365, Nov. 1950.

(9) Hoffman, W. S.: Clinical physiology of potassium. J. A. M. A. 144: 1157-1162, Dec. 2, 1950.

skeletal weakness with flaccid paralysis in seriously ill patients and electrocardiographic changes associated with a lowered broadened T wave, a lengthened QT interval, a depressed ST segment, and, finally, a U wave. An elevated serum potassium, on the other hand, inconstantly produces a flaccid paralysis of skeletal muscles and has its principal effects on heart muscle, the first electrocardiographic changes being a high peaked T wave manifested at levels of about 7.5 milliequivalents per liter, progressing to a broadened QRS complex at about 9.5 milliequivalents, and complete heart block with ventricular fibrillation at levels above 10 milliequivalents per liter *(10)*.

Clinically, in initial states of dehydration and acidosis, the serum potassium may be either normal or elevated. Only after rehydration does it begin to fall, dropping much more precipitously during recovery from diabetic acidosis than from other conditions. Until the child has been hydrated and is urinating freely, potassium should be withheld as a precaution against producing potassium intoxication. Thereafter he may be given potassium chloride solution in doses up to 20 cc. of a 1 percent solution per kilogram of body weight per day orally, or by drip hypodermoclysis if the oral route cannot be employed, but never intravenously.

SUMMARY

The importance of fluid balance in the young patient is of growing interest to the medical officer practicing military medicine because of the increased scope of dependent medical care. The plan of fluid therapy here presented has proved adequate for most pediatric problems. It provides for safety in the administration of optimal quantities of fluid and electrolytes.

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Myocardial Infarction Following Electric Shock

Lester E. Imboden, *Lieutenant, junior grade, MC, U. S. N. R. (1)*

Charles B. Newton, *Lieutenant Commander, MC, U. S. N. (1)*

ELECTRIC shock as a cause of myocardial infarction has been reported infrequently in the literature. Boas and Boas *(2)* refer without elaboration to electric shock as one of the causes of myocardial infarction. Vogt *(3)* reviewed the German literature on cardiac disturbances caused by electric shock and noted 2 patients with myocardial infarction. In 1 there was immediate substernal pain, but electrocardiographic evidence of infarction did not appear until 4 weeks later. In the other there was immediate chest pain and the patient died 24 hours following the shock. Autopsy revealed thrombosis in the left coronary artery. Koeppen *(4)* cited 93 patients with electrical accidents accompanied by cardiac symptoms in only 16 of whom were electrocardiographic abnormalities observed. The abnormalities were myocardial ischemia (T-wave inversion), auricular fibrillation, prolongation of the P-R interval, and the occurrence of premature ventricular beats. He applied the term "angina pectoris electrica" to the syndrome of recurrent chest pain following electric shock. The voltages to which these persons were subjected ranged generally from 220 to 50,000 and the current was of low frequency.

The psychiatric literature contains a large amount of material on the cardiac complications of electroshock therapy, the most frequent of which are minor arrhythmias, occasional serious arrhythmias, and cardiac dilatation *(5, 6)*. These rhythmic disturbances are believed to be caused by central vagal stimulation *(5)*. Kalinowsky and Hoch *(6)* stated

(1) United States Naval Hospital, San Diego, Calif.

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that low tension current in shock therapy would be dangerous because the heart might be included in the field of the current. Bellet et al. *(7)* noted inversion of T waves in leads 1 and 2 following electric shock therapy. He also found electrocardiographic changes indicating myocardial damage in two-thirds of 58 patients undergoing insulin shock treatment. Estes and Cleckley *(8)* reporting on electronarcosis noted the occurrence of gross arrhythmias, of acute congestive failure, and of a single case of severe congestive failure with transient electrocardiographic changes of myocardial ischemia. Plice and Pfister *(9)* studied the electrocardiographic abnormalities in 1,000 mentally ill patients and concluded that abnormal findings (especially T wave and S-T sector changes) which suggested myocardial damage often disappeared after electroshock therapy. Vast clinical experience has demonstrated the safety of high frequency electrical current as used in shock therapy.

The conduction of electric current through the body in patients with accidental shock is believed to be along the blood vessels *(10)*. High voltage alternating current may cause intense condensation of the surrounding tissue, thrombosis, and hemorrhage *(11)*. Gangrene may occur in the area through which the current has passed. This phenomenon is due to an extensive endarteritis involving the endothelium and subendothelial tissues of the large arteries with resulting thrombosis. The localization of changes indicates that the blood vessels offer the least resistance to conduction as the distant surrounding tissue is not injured *(12)*. It seems likely that myocardial infarction might be caused, in the manner outlined above, by direct injury to the coronary vessels by electric current, or in lower voltage current, by prolonged intense coronary spasm induced by electrical stimulation. Necropsy findings have also revealed petechial hemorrhages beneath the serous surfaces of the endocardium and pericardium *(13-15)*.

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CASE REPORT

A 34-year-old man was well until 1 week prior to admission on 9 April 1951 to the U. S. Naval Hospital, San Diego, Calif. On 2 April, while repairing an electric switch aboard ship, the fingers of his left hand were in contact with the metal portion of a screw driver when a friend diverted his attention and the screw driver made contact momentarily with a source of 440 volts of 60-cycle alternating current. He was stunned for about 30 seconds but did not lose consciousness. An observer stated that sparks flew from his mouth. Subsequent examination revealed that he was standing on a metal deck and that he was wearing a pair of regulation Navy shoes with worn rubber heels and leather soles that were in very poor condition. The right shoe contained an innersole but the left did not and was worn paper thin. Thus the path of least resistance was from the switch to screw driver to left arm to left foot and to the deck. The patient felt fairly well except for mild anginal symptoms, a vague sensation of fatigue, and residual numbness in the left arm and in the index and ring fingers of the left hand for about 4 days thereafter. He stated that on the third evening following the shock he did not feel entirely well during a birthday party given in his honor. About 36 hours after the electric shock, while taking a bath, he first noticed a severe pain in the left arm, left shoulder, and over the left chest. He felt his pulse at the wrist and noted that it was about 44.

The pain was practically gone the next morning but because of his not feeling entirely well he reported to sick bay where 2 ECG's were taken in the next 5 days. The second showed evidence of a posterior myocardial infarction and he was transferred to the hospital. On admission he appeared to be in no distress but was put to bed on routine coronary therapy. His liver was tender and was palpable about 2 finger-breadths below the costal margin on deep inspiration. No burned areas were noted on his hands or feet. His heart was not enlarged; the rhythm was regular; the sounds were distinct; and there were no murmurs. His blood pressure was 130/80 and his pulse was 80. His lungs were clear. His erythrocyte count was 4,800,000; his hemoglobin was 13.5 grams per 100 ml.; his leukocyte count was 6,000 with 80 percent polymorphonuclear cells, 2 percent band cells, and 18 percent lymphocytes. His sedimentation rate (Cutler) was 12. A roentgenogram of the chest was negative.

An ECG taken on 4 April (fig. 1) revealed a small Q_2 and Q_3 and a minute Q_{AVF}; T_{V1} was negative. An ECG taken on 9 April, revealed a moderate Q_2, Q_3, and Q_{AVF}. A negative T_3 and T_{AVF}; and a peaked T_{V2}, T_{V3}, T_{V4}, and T_{V5} (fig. 2). A diagnosis of posterior myocardial infarction was made. Subsequent ECG's on 10, 11, 16, 18, and 25 April and 2 and 23 May were unchanged except that T_2 became negative and coved on 16 April and remained so. The serial electrocardiographic

changes then began with an essentially normal tracing on 4 April and progressed through deepening Q waves and Pardee-type T waves in the posterior leads and began to return to the normal pattern 7 weeks later. Serial determinations of the sedimentation rate were: 30 on 11 April; 20 on 18 April; 18 on 1 May; and 22 on 23 May.

The patient was treated routinely at bed rest with anticoagulants and recovered uneventfully except for the development of premature systoles in the seventh week of his illness. The rhythm returned to normal with the administration of procaine amide hydrochloride orally.

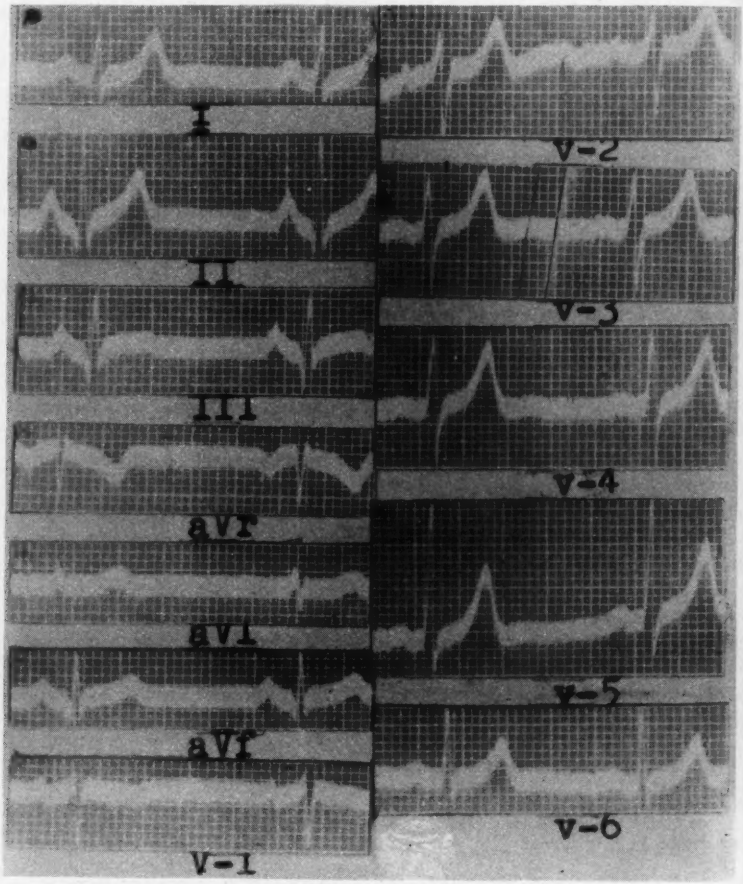

Figure 1. ECG's taken on 4 April showing small Q_2 and Q_3; a minute Q_{AVF} and a negative T_{V1}.

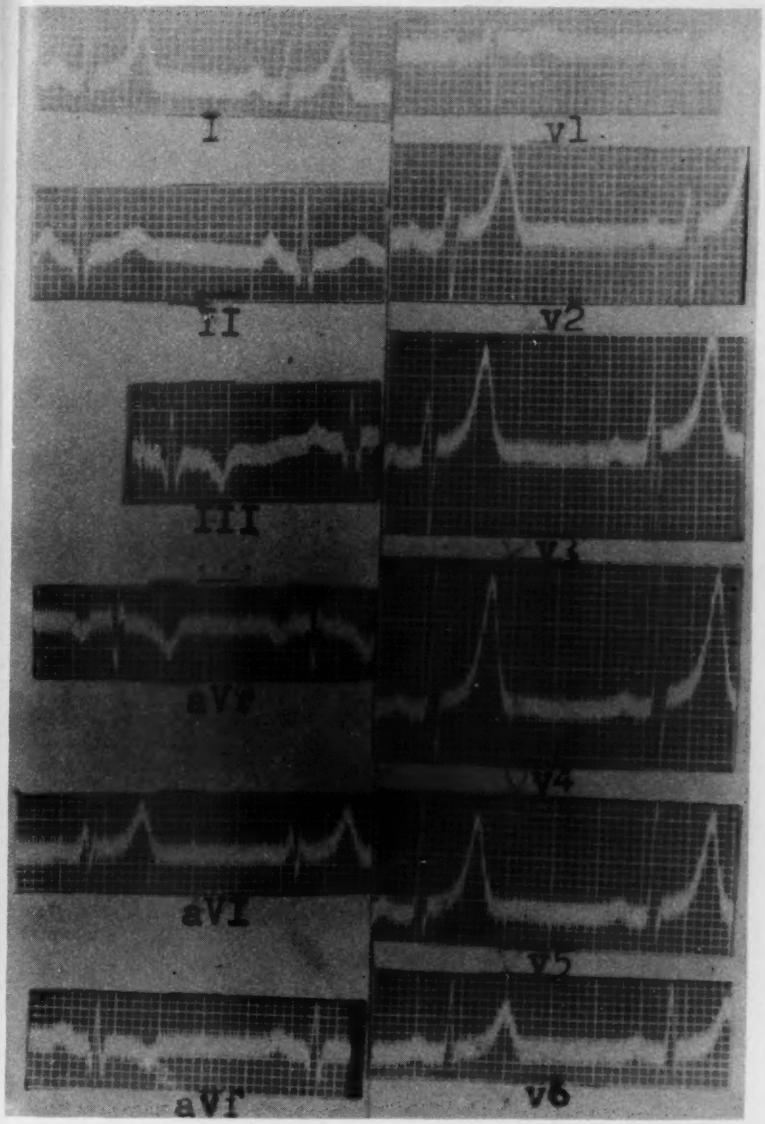

Figure 2. ECG's taken on 9 April showing moderate Q_2, Q_3, and Q_{AVF}; negative T_3 and T_{AVF}; and peaked T_{V2}, T_{V3}, T_{V4}, and T_{V5}.

COMMENT

In spite of the frequency of accidental electric shock in home and industry the complication of myocardial infarction is apparently rare. The patient here reported developed a myocardial infarction about 48 hours after a severe electric shock. This strongly suggests that the shock caused the infarction. Because the path of electric current through the body is along the blood vessels and severe blood vessel injury may result from electric shock it is not difficult to conceive of an electric current producing injury to or intense spasm of the coronary arteries when the heart is in the path of the current. The circumstances in this case suggested that the passage of the current was from the left hand through the body to the left foot which would quite likely include the heart in its course.

The use of high frequency current to the central nervous system in electric shock therapy has proved to be a relatively safe procedure and the cardiac complications that have occurred were undoubtedly on the basis of vagus effects on the rhythm or of overtaxing of a low cardiac reserve by suddenly increasing the circulatory requirements in convulsions. In accidental shock, however, when an electric current of low frequency passes through the body, there is a definite hazard of organic damage to the heart.